THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—41ST YEAR SYDNEY, SATURDAY, MARCH 13, 1954 No. 11

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— Page.

Observations Upon the Early Days of Anæsthesia in Australia, by Eric Gandevia 385
Fundamental Aspects of Allergy, by E. R. Trethewie 388
Psittacosis in Australia, by E. L. French, R. A. Joske, D. H. Bodycomb, E. F. Mackenzie and W. R. Rigg 392

REPORTS OF CASES—

A Case of Tracheo-Œsophageal Fistula, by Richard Flynn and Jeanne M. Collison 396
Bronchiectasis with Normal Plain X-Ray Appearances, by H. B. Harwood 397
Cancer of the Œsophagus Successfully Treated, by B. S. Hanson 400

REVIEWS—

History of the Second World War: United Kingdom Medical Series. Medical Research 401
History of the Second World War: United Kingdom Medical Series. The Emergency Medical Services 401
Modern Trends in Diagnostic Radiology 402

BOOKS RECEIVED 402

LEADING ARTICLES—

The Tenth Rheumatism Review 403

CURRENT COMMENT—

A Double Centenary 404
The Dermal Mirror 405
Sterilization Procedures in Hospitals 406
Iproniazid in the Treatment of Bone and Joint Tuberculosis 406
The Prevention of Airsickness 407

ABSTRACTS FROM MEDICAL LITERATURE—

Pædiatrics 408
Orthopædic Surgery 409

SPECIAL ARTICLE—

Notes on Traumatic Surgery in Korea 410

MEDICAL SOCIETIES— Page.

Melbourne Pædiatric Society *413*

OUT OF THE PAST 416

SPECIAL CORRESPONDENCE—

New Zealand Letter 416

CORRESPONDENCE—

The College of General Practitioners 417
Perforation of the Œsophagus and Rupture of the Aorta due to a Piece of Fly-Wire 417
The Treatment of Acute Poliomyelitis 417

OBITUARY—

Alexander Juett 417
Alexander Hammett Marks 418

NAVAL, MILITARY AND AIR FORCE—

Appointments 418

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 419

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—

Queensland 419
Tasmania 419

POST-GRADUATE WORK—

The Post-Graduate Committee in Medicine in the University of Sydney 420

NOTICE—

Stolen Electrocardiograph 420

DEATHS 420

MEDICAL APPOINTMENTS 420

NOMINATIONS AND ELECTIONS 420

DIARY FOR THE MONTH 420

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 420

EDITORIAL NOTICES 420

OBSERVATIONS UPON THE EARLY DAYS OF ANÆSTHESIA IN AUSTRALIA.[1]

By ERIC GANDEVIA,
Retiring President, Australian Society of Anæsthetists, Melbourne.

If men will tend to observe, they shall find much worthy to observe.

—FRANCIS BACON, "The Advancement of Learning".

VERY little has been published on the history of anæsthesia in this country, and none of it, to my knowledge, has been written by an anæsthetist. I feel that the Australian Society of Anæsthetists should encourage members to interest themselves in this important subject, and with that object in view the State sections might with advantage devote one meeting each year to a paper on the historical aspect of anæsthesia. Three articles provided me with most of the material for this survey: "The Introduction of Surgical Anæsthesia in Van Diemen's Land", by W. E. L. H. Crowther, read at the Centenary of the Introduction of Surgical Anæsthesia to Australia; "An Essay Relating Chiefly to Anæsthetics and their Introduction into Australia", by N. J. Dunlop (1927); and an excellent article by W. L. Potter (1938), "Anæsthetics in Australia in the Early Days".

It is not inappropriate that this address should be delivered in Adelaide, for some of the most notable contributions to the early Australian literature came from J. D. Thomas, W. E. Jay, and others of the Adelaide Hospital, who established a tradition so worthily carried on by Gilbert Brown.

On June 7, 1847, ether was administered to two patients at Launceston by Dr. W. R. Pugh. The first patient was suffering from a painful disease of the lower jaw; the second from a cataract. This was the first occasion that general anæsthesia was employed in Australia. It is worthy of note that these two administrations took place only eight months after the first use of ether by Morton in Boston, Massachusetts.

The lay Press at that time was the chief means of disseminating medical information, not only to the general public, but also to members of the medical profession—a practice which perhaps is tending to recur. The Launceston *Examiner* had, on June 2, reported the use of ether in London five and a half months before, and on June 9 its issue contained a detailed and accurate report of the first two administrations by Dr. Pugh, together with a description of the apparatus employed. This Pugh had constructed on the same lines as Nooth's carbon dioxide apparatus, which was described in the *Illustrated London News*. How would one of us fare if asked to induce anæsthesia with this machine?

[1] Read at a meeting of the Australian Society of Anæsthetists on April 20, 1953, at Adelaide.

> It consisted of two glass vessels, one placed on the other and both containing pieces of sponge saturated with ether. Connected with the lower vessel is a flexible tube provided with a stopcock, etc., terminating in a pipe which is placed in the patient's mouth. The upper vessel is fitted with a glass stopper which is removed when the patient inhales, so that the air he breathes passes through the saturated sponges and thus becomes saturated with the ether they contain. The nostrils being compressed, the process of breathing is precisely as if a person inhaled and expressed the atmosphere through a tobacco pipe.

No attempt was made to maintain anæsthesia for any length of time, for it was in fact a "single-dose" method suitable only for short operations.

If we bear in mind the difficulty all of us have experienced in anæsthetizing robust patients with ether alone, even with the help of premedication and modern apparatus, it is not surprising that early enthusiasm gave way to doubt. Ten days later the *Examiner* published the following letter from Dr. Pugh.

> Sir,—In a recent number of your journal, you noticed two surgical operations performed by me at St. John's Hospital, in which the recently-discovered agent, Sulphuric Ether, was employed to render the parties unconscious and insensible to pain. The result of this trial was so far satisfactory as to lead me to hope that in future surgery would cease to be looked on with that dread which had been from the earliest times associated with its proceedings. I regret that my further acquaintance with this much lauded agent has tended materially to lessen its value in my estimation. I have found that its effects on individuals are as variable as the constitutions of those by whom it is inhaled. Persons weakened by previous disease are quickly brought within its influence and those of feeble frame offer but little resistance to its action. Not so, however, with the hearty and robust; the stage of depression in such is deferred and all the excitement common to excessive drunkenness is presented, the features become bloated and livid, the eyes protruded and suffused, and the disposition of the party when inebriated is fully developed, and these effects are not of the transitory character they are reputed to possess. The brain remains confused and the stupor of the drunkard is experienced for long subsequent to the application which occasioned it. I have seen sufficient to create alarm in my mind and therefore would caution the profession against the applying of the inhalation of Ether without special reference to the individual case. The remedy, like its twin brother, nitrous oxide gas, is, I feel assured, destined to enjoy a short lived notoriety. Their objectionable qualities are of a similar character, having alike a similar tendency to produce congestion of the brain, and therefore those circumstances which occasioned the removal of nitrous oxide from the list of remedial agents, in which for a short time it held a high position, will in all probability deprive the sulphuric ether of portion of the credit it has recently received.
>
> I have the honour to be,
> Yours most obediently,
> W. R. PUGH, M.D.

In June, 1847, in an editorial in the short-lived *Australian Medical Journal*, the following statement was made:

> The plan now proposed and which has been imported to Great Britain from America is to intoxicate the patient by causing him to inhale the vapour of Sulphuric Ether. Now, although the inebriation produced is evanescent, it is still inebriation and it does not appear to us of much consequence if the operatee must be drunk what the intoxicating medium may be. We have no hesitation in predicting for this process a transient popularity, it will have its day, ultimately to be abandoned as useless or injurious.

Finally, about this time, a medical practitioner wrote to a London paper to the effect that inhalation of ether seriously affected the blood and tended to produce pulmonary tuberculosis.

The critical attitude of those early pioneers seems to be in contrast to the enthusiasm shown for new anæsthetic drugs by the profession today, and so well illustrated by the history of cyclopropane. However, one member of the profession did not share these pessimistic views. He was Dr. D. J. Thomas, who on August 2, 1847, performed the first surgical operation under ether at Port Phillip. He also had the distinction of reading the first scientific paper before a medical society in Victoria, the title being "On the Inhalation of the Vapour of Æther, with Cases". This paper was rediscovered by Dr. G. T. Howard. The *Australian Medical Journal* became extinct before the paper was published, but the manuscript is in the library of the British Medical Association in Melbourne and was reprinted in full in the *Royal Melbourne Hospital Clinical Reports* in 1933. Dr. Howard describes Dr. Thomas as "a genial Welshman, a Member of the Royal College of Physicians and a Licentiate of the Society of Apothecaries (1838), a delightful combination of Puck, Peter Pan, Fluellen and shrewd alert efficient leader of the profession". Thomas disagreed with the remarks of the editor of the *Australian Medical Journal* and others that ether would be short-lived. He said:

> Who would give ether vapour to a patient in whose lungs there existed a deposit of tuberculous material? I would be sorry to etherize such a patient. Neither would I recommend a long walk or a shower every day. From all I have seen and from cases I have read in which it has proved injurious, I am convinced they are the result of improper administration.

Following the use of general anæsthesia one would have expected surgical operations to show a progressive increase in numbers; but, as was pointed out by Potter, the reverse was the case. Potter stated that after early cases had been reported there were 21 operations under ether in ten months, 13 during the first five months and eight during the next five months. A similar but more pronounced falling-off in the use of chloroform occurred after its introduction. There were 24 operations in ten months, 20 during the first five months and four during the next five months. These figures are derived from a survey of newspaper reports (1847 to 1849) made by Dr. Potter. However, it is doubtful whether they reflect the position accurately, as newspaper interest in the innovation may have subsided. It would be of value to examine some hospital statistics of the period. The falling-off in the case of ether was ascribed to (i) Dr. Pugh's letter to the *Examiner*, (ii) the editorial in the *Australian Medical Journal*, and (iii) the letter by a medical practitioner to a London paper. The decrease in the use of chloroform may have been due to the high mortality. Later still, there was a temporary abandonment of ether because of many supposed advantages of chloroform.

Chloroform was first used in Australia on April 11, 1848, by Dr. McEwan, of Sydney, who was also the first New South Welshman to use ether. The nature of the operation was not specified, but it was said to be "most tedious, difficult and hitherto excruciating". This operation took place only five months after the introduction of chloroform by Simpson. I have been unable to find a record of the first administration of chloroform in Melbourne, but the Melbourne *Argus* of May 26, 1848—that is, about six weeks after McEwan used chloroform in Sydney—contained the following paragraph:

> Chloroform. We have much satisfaction in drawing attention to a notice in another column intimating that Mr. Hood, the druggist, has succeeded in the preparation of chloroform, the invaluable agent now used by the medical profession for doing away with all sense of pain in submitting to surgical operations.

In another column appeared the following:

> Mr. Hood begs to intimate that he has succeeded in preparing chloroform in a pure state and that he is enabled to supply it at a moderate price. Collins Street, opposite Commercial Inn.

It is interesting to note that in 1852 the famous J. G. Beaney was working as an assistant in Thomas Hood's shop. In Beaney's "Original Contributions to Conservative Surgery" (1859), the first book on surgery produced in Victoria, a chapter is devoted to anæsthesia, while a monograph entitled "The Necessity for Anæsthetics to Alleviate Human Suffering" appeared under his name in the *Medical and Surgical Review of Australasia* in 1873. Despite his apparently authoritative position, we find that Beaney, when confronted with an anæsthetic emergency, favoured brandy given by mouth as a vital resuscitative measure (B. Gandevia, 1952).

From 1852, and for some years thereafter, chloroform figured prominently in the journals and ether scarcely at all. There were two reasons for this: (i) chloroform had largely superseded ether; (ii) deaths from chloroform were frequently reported. This was borne out by Dr. J. Davies Thomas (not to be confused with Dr. D. J. Thomas, mentioned earlier), formerly chloroformist at University College Hospital, London, who in May, 1875, when resident surgeon at Clunes Hospital, read a paper entitled "A Consideration of Respective Merits of Chloroform and Ether". In November, 1875, when senior house surgeon at Adelaide Hospital, he contributed an article on ether and chloroform as anæsthetics. Thomas explained that, in the former paper, he had endeavoured to show the reasons which had led him, as they had many others, to question the validity of the general preference of British surgeons for chloroform. He was able to collect reports of 177 deaths from chloroform, but only 45 from ether. The estimated deaths from ether were 1 in 23,000 and from chloroform 1 in 2500. The account of the first death under ether was obtained by Thomas from Dr. Snow's book on anæsthetics. This occurred on July 10, 1847, and Thomas makes the following statement:

> The patient was simply asphyxiated and no proper attempt at resuscitation made . . . but nowadays I cannot conceive a qualified man allowing a patient to die in this way.

Could Thomas review a series of notifiable deaths today he would doubtless be disappointed to note that asphyxial deaths are still occurring. Detailed accounts of death during ether anæsthesia indicate that they were nearly all asphyxial, even as they are today. Thomas stressed the fact that not all deaths during anæsthesia were due to the action of the anæsthetic, but that some were caused by "accidental epi-phenomena". He mentioned four: (i) Terror or mental anxiety may cause fatal syncope. (ii) It may happen that the predestined "supreme moment" (that is, death) may coincide with the time of administration of the anæsthetic. (iii) The physical state of the patient is sometimes such that the most trifling additional injury may cause fatal syncope. (iv) Certain operative procedures tend to cause syncope and, although anæsthesia diminishes shock, it does not altogether abolish it—for example, when the spermatic cord is being cut.

This is of interest, as it seems to foreshadow Crile's work of about thirty years later.

In 1876 Thomas again wrote on chloroform *versus* ether and quoted twenty rules for the administration of the latter from the work of Dr. Bigelow, of Boston. All of them, with slight modification, are applicable to the administration of ether today. Of special interest are the following:

> 1. If you can devote more time to the process [of induction] the resistance will be less.
>
> 2. If the patient is rigid or livid give him air.
>
> 3. If his glottis contracts (i.e., laryngeal stridor) give him air.
>
> 4. If he breathes badly put the finger inside the cheek to admit air over the base of the tongue.
>
> 5. In an operation about the nose and throat, if such an operation promises much blood, have a tracheotomy tube ready, or insert the tube before operation and put a sponge in the pharynx.
>
> 6. Although there is less nausea with an empty stomach, it is not well to starve a patient about to encounter a protracted operation.

As was mentioned previously, the high mortality under chloroform gave occasion for frequent discussion in the journals. The conclusions arrived at during the first ten years, although the knowledge was empirical, were amazingly accurate. In 1856 the following statements were made: (i) In nearly every case of death during chloroform anæsthesia the patient died during induction. (ii) Autopsy disclosed nothing to account for death. (This still holds good for deaths during the administration of other toxic agents—for example, "Pentothal", spinal analgesics.) (iii) Dr. Snow, who wrote a book on anæsthesia in 1847, observed that deaths during chloroform anæsthesia were occasioned by sudden arrest of the heart, but that if the vapour was inhaled in a regular and gradual manner it would be impossible to paralyse the heart by its direct influence.

If every anæsthetic agent was administered in the "regular and gradual manner" which Snow advised nearly a century ago, fewer accidents would occur with the potent drugs available today.

With the high mortality of chloroform, methods of resuscitation were frequently debated. In the main, the basic principles adopted during the first fifteen years of general anæsthesia persisted until only a few years ago. Some were tried and found wanting. For example, on one occasion 20 ounces of blood were removed from a distended jugular vein, doubtless distended because of respiratory obstruction. Dr. Maloney, during a discussion on chloroform, said that vigorous bumping of the head against the table was practised by Mr. T. N. Fitzgerald and alarming symptoms were quickly banished in this way. Ammonia had been used orally as a stimulant in cases of poisoning; but Professor Halford, professor of anatomy and pathology at the University of Melbourne, to use his own words, "determined to follow up the poison as quickly as possible by injecting directly into the blood, thus avoiding rejection and decomposition by the stomach and intestine and in the case of ammonia reaching the heart without obstruction by or danger to the respiratory passages". Professor Halford was reported to have resuscitated dogs from a state of anæsthesia in which the heart's action had actually ceased, by means of the intravenous injection of ammonia. Hence for some years this was used as a routine resuscitatory measure. One issue of the *Australian Medical Journal* was devoted to the cure of snake-bite by this means. Forty-two cases were reported in full, with only one death. The preparation used was *Liquor Ammonii* (British Pharmacopœia), and about 1·5 drachms, diluted with water, were injected. Probably this method was just as effective for cardiac arrest as the intravenous injection of "Coramine" of recent years.

Details of the usual methods of resuscitation at that time are well described by Dr. W. E. Jay, senior house surgeon of the Adelaide Hospital. In February, 1875, Dr. Jay described a death due to the administration of chloroform, "to which hitherto we have had immunity". The patient's death seemed to be instantaneous and Dr. Jay then described the measures employed to resuscitate him:

> Every door and window was opened, the tongue pulled forward, cold water dashed on face and abdomen. Artificial respiration resorted to, galvanism too, along course of phrenic nerve and over heart—Nelaton's method based on supposed anæmia of brain was followed, namely:—head and body inverted, legs thrown over shoulders of one of the house surgeons while others continued artificial respiration. This was persevered with for three-quarters of an hour without getting symptoms of returning consciousness.

He concludes:

> My object in sending above case is not merely that all such should be known to the profession, but, when such a lamentable occurrence as death from chloroform takes place, it may be published and quoted to serve as a guide to juries and to inform the public how impossible it is to prevent such accidents notwithstanding the greatest caution that can be used.

The use of galvanism is of special interest. Sansom in 1866 described the use of galvanism over the phrenic nerve as a means of resuscitating patients who had collapsed under anæsthesia. Sarnoff in America recently developed it as a means of artificial respiration. Recently, in the *Royal Melbourne Hospital Clinical Reports*, there was the report of an investigation undertaken to confirm the claim that adequate ventilation was produced by unilateral phrenic nerve stimulation (B. Gandevia, Ross and Bolton, 1952). The Director of Anæsthesia, having offered himself as a guinea-pig, is said to have been impressed with the possibilities of this method for resuscitating patients who have collapsed during anæsthesia.

It would be invidious to compare the results of the first generation of anæsthetists with the results of our own generation, in view of the vast advances in physiological and pharmacological knowledge and the development of

new techniques. Nevertheless, it would be most unwise to underrate the skill of the early exponents of the anæsthetic art (for it is no less an art with a "rag and bottle" than it is with a closed circuit and controlled respiration), who, as we have seen, were able to formulate the essential principles of safe anæsthetic administration on a basis of empirical observation. Although isolated by a voyage of perhaps months from the chief medical centres of the world, they were unafraid to put a new idea to the test as soon as reports appeared of its use overseas. Thrust upon their own resources to a considerable extent, they were able to appreciate the difficulties and dangers of, and the indications and contraindications for, each of the available agents.

The accuracy of their conclusions is evidenced by the fact that, although ignorant of "ventricular fibrillation", they realized the direct action of chloroform on the heart and the risk of sudden death, especially during the induction stage. They also realized that toxic agents were safer if given diluted and in gradually increasing doses; that most deaths during ether were asphyxial; perhaps, too, they anticipated an application of electricity to medicine. Other examples could be quoted. If we have lessons to learn from the anæsthetists of yesteryear, then I suggest that they are the following: (i) the value of careful, accurate clinical observation; (ii) the value of independence of thought and practice without recourse to reliance on the reports of others; (iii) the value of a critical approach to a new method, drug or appliance.

I would suggest that the future of our specialty lies ultimately more in the application of these lessons than in the specialized researches of the biochemist, the pharmacologist and the physiologist. While we are dependent upon these for our technical advances, the responsibility for their application rests with us. In this regard the approach of our predecessors may be commended. They followed another of Bacon's precepts:

> If a man will begin with certainties, he shall end in doubts; but if he will be content to begin with doubts, he shall end in certainties.

References.

CROWTHER, W. E. L. H. (1947), "The Introduction of Surgical Anæsthesia in Van Diemen's Land", M. J. AUSTRALIA, 2: 561.

DUNLOP, N. J. (1927), "An Essay Relating Chiefly to Anæsthetics and their Introduction to Australia and Tasmania", M. J. AUSTRALIA, 1: 285.

GANDEVIA, B. (1952), "Death under Anæsthesia: Mr. J. G. Beaney's Case", M. J. AUSTRALIA, 1: 291.

GANDEVIA, B., ROSS, M., and BOLTON, J. H. (1952), "The Effects of Electro-Phrenic Respiration in Normal Subjects", *Roy. Melbourne Hosp. Clin. Rep.*, 22: 10.

HOWARD, G. T. (1933), "The First Scientific Paper Read before a Medical Society in Victoria", *Roy. Melbourne Hosp. Clin. Rep.*, 4: 67.

JAY, W. E. (1875), "A Case of Death from Chloroform", *Australian M. J.*, 20: 47.

POTTER, W. L. (1938), "Anæsthetics in Australia in the Early Days", M. J. AUSTRALIA, 2: 940.

THOMAS, D. J. (1847), "On the Inhalation of the Vapour of Æther, with Cases", reprinted in *Roy. Melbourne Hosp. Clin. Rep.* (1933), 4: 68.

THOMAS, J. DAVIES (1875), "A Consideration of the Respective Merits of Chloroform and Vinic Ether as General Anæsthetics", *Australian M. J.*, 20: 170.

THOMAS, J. DAVIES (1875), "Ether and Chloroform as Anæsthetics", *Australian M. J.*, 20: 375.

FUNDAMENTAL ASPECTS OF ALLERGY.[1]

By E. R. TRETHEWIE,

Department of Physiology, University of Melbourne.

I AM honoured that you have invited me to address you on the subject of allergy. I have been asked to speak largely on work with which I have been associated personally, and that I consider "fundamental aspects". This, within limits, I shall endeavour to do.

[1] Read at a meeting of the Section for the Study of Allergic Diseases of the Victorian Branch of the British Medical Association on November 5, 1953.

There are two major problems concerned: (i) the pharmacology of the process involved; (ii) the reason for the development of the sensitized state.

Rather loosely we group the state of allergy with that of anaphylaxis, though there are fundamental differences. In anaphylaxis following the exhibition of a pharmacologically moderate (though actually very tiny) dose of antigen, desensitization is then complete. In allergy the sensitized state remains or readily recurs, apparently *de novo*. It may be in the latter instance that the nuclei of the cells have "learned" to develop certain biochemical phenomena. This process may then recur in the absence of the sensitizing agent.

The first clear-cut evidence that histamine was indeed responsible for many of the phenomena of anaphylaxis was presented by Bartosch, Feldberg and Nagel in 1932. At the same time, and independently in America, Dragstedt and Gebauer-Fuelnegg (1932) showed that histamine being derived from the liver appeared in thoracic duct lymph in the dog in anaphylaxis. This was the continuation of the work of Dale on the subject, who many years previously had produced indirect evidence that histamine was probably liberated. It was then believed that the combination of antigen with antibody in the cell (though probably on the surface) caused profound alteration of permeability of the cell membrane and the release of preformed histamine. This was released largely into the tissue spaces. That other substances were released was shown by Dr. C. H. Kellaway and myself in 1940, when we demonstrated the release of S.R.S., a slow-reacting muscle-stimulating substance, whose release was a little more prolonged than that of histamine. In Canada, Jaques and Waters (1941) showed that heparin was released in the anaphylactic reaction. Probably many other substances are liberated too.

That the substance histamine was indeed released was disputed from time to time, although the evidence was practically conclusive. More recently with the aid of antihistamines I have been able to show (Trethewie, 1951*a*) that in addition to multiple assay indicating its nature there is a strictly parallel differential inhibition by antihistamine for both histamine and the histamine-like substance released in anaphylaxis. No one has doubted seriously in the past ten years that histamine itself is the substance released.

Now during this period one aspect has been overlooked. The significance of the formation of a compound of the nature of anaphylotoxin—that is, a substance formed between say peptone and plasma, acting as a damaging agent like that of anaphylaxis, has been relegated to the background. Much literature had been written on this subject in the past. Rocha e Silva has renewed interest in this phenomenon. Ungar and Mist (1949) have shown that fibrinolysin is formed this way. Feldberg and O'Connor (1937) showed that peptone caused the release of histamine from the Tyrode solution perfused (blood free) lung of the guinea-pig, and I attempted to show (Trethewie, 1938) in the same laboratory the following year that histamine was released by peptone from the Tyrode solution perfused liver of the dog. The experiments, eight in all, completely failed to show the release of histamine. This puzzled us greatly. Again, at about that time, Dr. Kellaway and I (Kellaway and Trethewie, 1939) attempted to show the release of histamine in anaphylaxis from the Tyrode solution perfused liver of the dog, and again the experiments were completely unsuccessful. The explanation of this was forthcoming from the work of Rocha e Silva and Grana, who showed in 1946 that peptone did not cause the release of histamine from the blood-free perfused liver, but that when the organ was perfused with blood (Rocha e Silva *et alii*, 1947) histamine was indeed liberated. This, then, explains why Dragstedt and Gebauer-Fuelnegg were able to demonstrate the release of histamine from the liver of the intact dog, while Dr. Kellaway and I were unsuccessful with the isolated organ perfused with Tyrode solution. The significance of this was pointed out by Dr. Kellaway in a recent lecture (Kellaway, 1947) in Edinburgh. It would appear that, as was maintained by Rocha e Silva, the formed elements of the blood and

ILLUSTRATIONS TO THE ARTICLE BY E. L. FRENCH, R. A. JOSKE, D. H. BODYCOMB, E. F. MACKENZIE AND W. R. RIGG.

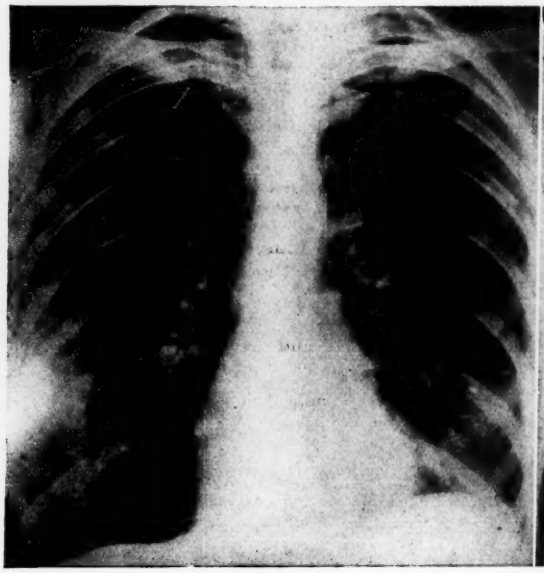

FIGURE II.

Radiograph of the chest in a case of psittacosis (Case IV) before commencement of therapy. There are hazy "ground-glass" opacities in the right mid-zone and at the base of the left lung.

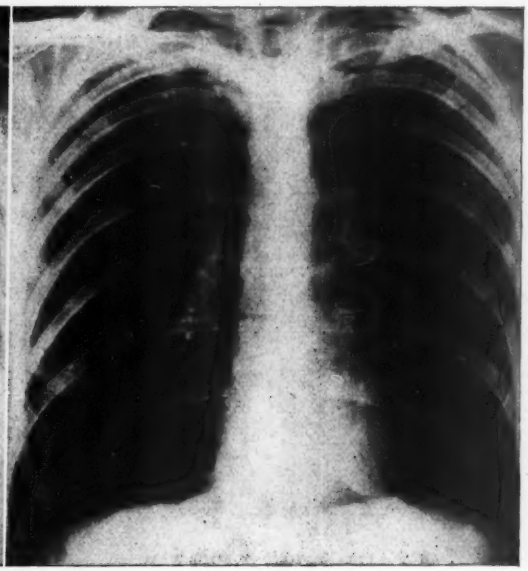

FIGURE III.

Radiograph of the chest in the same case (Case IV) after treatment with aureomycin. Some faint opacity persists in the right mid-zone and at the base of the left lung, although clinical examination revealed no abnormality and the patient had returned to work.

ILLUSTRATIONS TO THE ARTICLE BY RICHARD FLYNN AND JEANNE M. COLLISON.

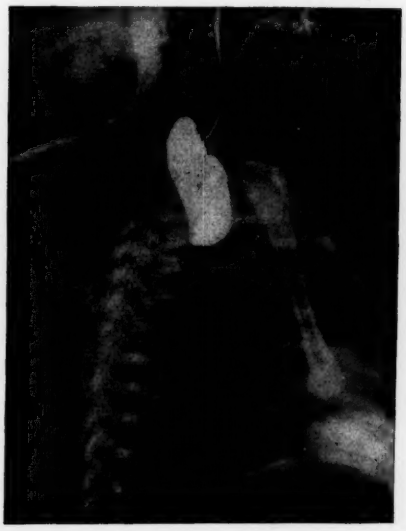

FIGURE I.

FIGURE II.

ILLUSTRATIONS TO THE ARTICLE BY H. B. HARWOOD.

Figure I.

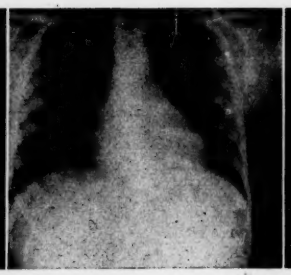

Figure II.

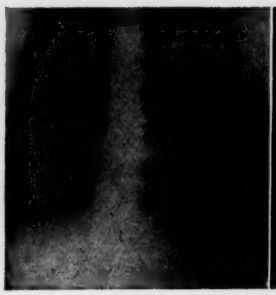

Figure III.

Figure IV.

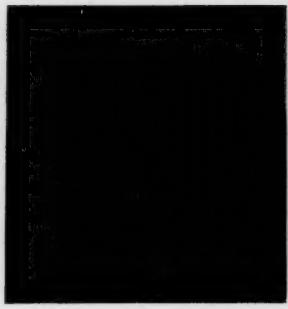

Figure V.

Figure VI.

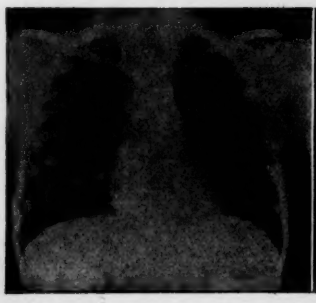

Figure VII.

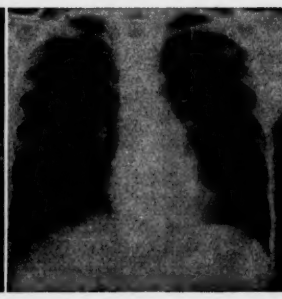

Figure VIII.

protein take part in the anaphylactic reaction and cause the release of considerable amounts of histamine. The same phenomenon occurs in peptone shock. But further phenomena are introduced when the blood-perfused organ is considered. Anticoagulant other than heparin is liberated by the blood-perfused organ (Trethewie, 1945*a* and *b*). In 1949, Mr. Day and I (Trethewie and Day, 1949) were able to show that while black snake venom caused the release of histamine from the blood-perfused liver of the dog, as it did in the case of the Tyrode solution perfused liver (Trethewie, 1939), there was released in addition in the case of the blood-perfused organ a conjugate of histamine, and an inhibitor substance was detected. The conjugate, as had been shown in urine samples (Anrep *et alii*, 1944), could be split up by acid hydrolysis (Trethewie and Day, 1949), free histamine being liberated. A little later, and independently, Tabor and Mosettig (1949) showed that conjugation of histamine occurred when liver powder was mixed with co-enzyme A and histamine. Then Miss Gaffney and I (Trethewie and Gaffney, 1951) showed that histamine was conjugated following the intraportal injection of free histamine into the blood perfused liver of the dog. Differential inhibition with antihistamine was used in the assays to confirm the release of histamine following hydrolysis of conjugate. The activity of the liver in regard to conjugation, actually acetylation, is very rapid. In the two or three minutes during which the perfused blood is in contact with the liver, in some instances we found 80% of conjugated histamine occurring.

Now this feature of conjugation of histamine may be important in reducing toxic effects of histamine, although in general histamine is largely liberated into the tissue spaces and little reaches the general circulation. However, that the blood level may be important is well shown in the anaphylaxis experiments of Dragstedt and Gebauer-Fuelnegg, who found reasonable amounts of histamine in thoracic duct lymph, which presumably soon found its way into the general circulation. Destruction of histamine by histaminase can occur, but it is slow. Kellaway and I (1940) were not impressed by its activity, and Blaschko was in agreement, though more recently the significance of histaminase has been remooted. On the other hand, the extreme rapidity of the acetylation mechanism with the living blood perfused liver suggests that this detoxicating mechanism would be more valuable biologically, as indeed are many other forms of acetylation encompassed by the liver.

Another feature which may explain varying degrees of effect of histamine release is related to a phenomenon which I have termed "non-specific immunity" (Trethewie, 1949), whereby past experience of histamine liberation influences the release of histamine by other agents. There are two phenomena involved here. In general, we find on the one hand that histamine is liberated more readily once liberation has taken place from a preceding injury. This is a general biological principle, also recognized in the case of digestion. It would appear that it is equally important in tissue injury. An interesting point, however, is that the lethal dose of histamine after previous injury actually rises. This would indicate that preceding experience of injury makes the organism more able to withstand the toxic effects of histamine. It suggests that preceding experience of a histamine-liberating agent may make the local reaction more intense with repeated injury, but that the organism as a whole is more able to withstand systemic effects of histamine by diminished toxicity for the whole animal. It is not inconceivable that such phenomena may be important in such an infectious disease as tuberculosis, to which there is frequently superadded an important allergic process. Further, Mr. Day and I (Trethewie and Day, 1949) have shown that in very young animals more histamine is released relative to the size of the animal by an injurious agent, and the lethal dose of histamine in the young animal is less relative to size. It is not unlikely that the same phenomenon applies to man as to the guinea-pig, as both are "allergic" creatures with a moderate histamine content of the lung, and in each the lung forms the shock organ in anaphylaxis. It is well recognized that in man many allergic manifestations of childhood recover spontaneously or diminish with time.

Not only does previous experience of injurious agents influence the responsiveness to histamine, which is undoubtedly important in the allergic process, but also the activity of the endocrines is likewise operative. Miss Gaffney and I (Trethewie and Gaffney, 1949) showed that serum gonadotropic hormone affected the histamine content of the lung and increased the lethal dose of histamine. In general it is recognized that the higher the initial content of the lung in histamine, the greater is the release of histamine from a standard injury (Trethewie, 1939). We then showed (Trethewie and Gaffney, 1950) that the gonadal hormones increased the histamine content of the lung, and that the lethal dose of histamine—that is, the sensitivity to histamine—was depressed. More recently the pronounced effect of ACTH on the allergic response has been demonstrated; for example, in *status asthmaticus* one of the more certain treatments is the slow intravenous injection of ACTH. This therapy is believed to reduce the sensitive state, and the mechanism may be related to the foregoing findings.

The effect of heparin in reducing the release of histamine in tissue injury has also been pointed out by Rocha e Silva (1952), and it may take part in reducing the cellular interaction by preservation of platelets. However, that there is a specific cellular inhibition of heparin is shown by the experiments of Mr. Day and myself (Trethewie and Day, 1949), in which we showed that heparin reduced the release of potassium from the isolated perfused (blood-free) liver. I have further pointed out (Trethewie, 1952) that this effect of heparin might explain the pain-relieving effect of heparin in coronary infarction, for there has been evidence to suggest that the release of potassium might take part in the production of pain in that condition. However, not only does heparin reduce the release of potassium, but release of histamine, and probably of other tissue-injurious substances released in tissue injury, is also inhibited.

It would therefore seem that histamine is an important pharmacologically active substance in anaphylaxis, and that there are numerous factors influencing the toxicity of histamine. In some instances the importance of histamine is almost paramount—for example, in *erythema multiforme* (often from drug sensitization), in which we find rapid amelioration of symptoms following the taking of antihistaminic drugs. In asthma the pattern is more complicated. It has already been indicated that substances other than histamine are liberated in the allergic state, and in view of the not universal value of antihistamines in asthma many regard the problem as more concerned with other factors. Undoubtedly physical action of muscles is important, particularly that of the diaphragm, and there may be paradoxical breathing; but it may be that improvement following physiotherapy is due to the overcoming, by more powerful inspiration, of bronchial constriction produced by histamine release. Further, all actions of histamine are not inhibited by antihistamine; for example, the secretion of acid in the stomach in response to histamine injection is not inhibited by antihistamines generally, though it has been claimed that a recently introduced compound (phenyl-methyl-tetrahydroazofluorene) does this.

Psychogenic stimuli undoubtedly also play a large part in many of the allergic phenomena, such as asthma; but in my experience they form more a psychological overlay, which largely disappears, often gradually, when the fundamental phenomena are put to rights. On the other hand, the danger is that with long-continued illness the psychological overlay can, as with a conditioned response, largely replace the original exciting cause, and this requires intelligent handling. The fact that there is evidence too for histaminergic nerves in the lungs opens up wide possibilities in this regard. "Bronchitis" also plays a role.

Concerning the mechanism involved, therefore, we may regard it as follows. After the exhibition of antigen the organism develops an abnormal immune response; perhaps in the guinea-pig circulating antibody attaches itself more firmly to the lung "parenchyma" or is not washed out so

readily. When the antigen meets with the antibody on the cell surface, profound changes occur and preformed H substances are released. These are histamine, S.R.S., heparin and probably many other substances (Figure I). In addition, in some way the cellular elements of the blood take part in the reaction too; according to Rocha e Silva and his co-workers, rapid disintegration of micro-thrombi of platelet and leucocyte also occurs, and the foregoing and probably other active substances are released in the dramatic reaction. In the dog this reaction is necessary to allow the cellular reaction in the liver referred to above to occur. In the guinea-pig this is apparently not essential for this reaction. Man is like the guinea-pig in that the shock organ is the lung and not the liver, and so perhaps

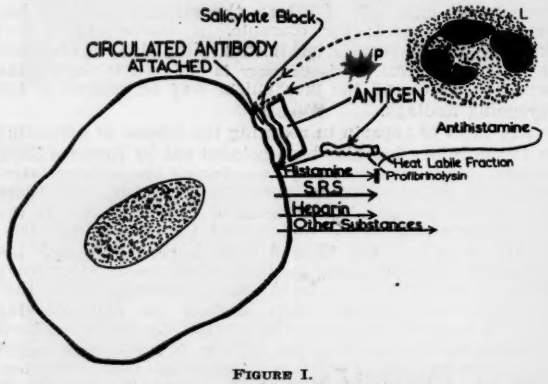

FIGURE I.

Schema of anaphylactic reaction indicating the part played by antigen, antibody, anaphylotoxin, formed elements and salicylate block. Antibody is attached to the surface of the cell (especially firmly in the guinea-pig), and the platelet (P) and leucocyte (L) take part in the reaction with antigen in damaging the cell. Associated with this reaction antigen reacts with profibrinolysin (in the sensitized animal) to produce toxic substance.

in man too the reaction follows the lines of that in the guinea-pig. Removal of these cellular elements, as with nitrogen mustard, may inhibit the development in the dog, though one might not expect this to be completely inhibitory in man if our supposition was correct. On the other hand, Rocha e Silva has shown that citrate can protect the skin from the development of allergic response in man, and indicates that his experimental findings support the mechanism as a rational therapeutic procedure. One must also assume that previous experience, the activity of the endocrines and histamine detoxicating mechanisms in the subject probably influence the degree of damage in anaphylaxis.

With regard to the reason for the development of the allergic state, little is known. Owing to the frequent occurrence in young people with "chemical" allergy (pollens, chemical substances *et cetera*) of achlorhydria (Trethewie, 1947), Day and I attempted to determine whether there was a reduced carbonic anhydrase activity of the blood of these subjects which might suggest a biochemical difference in "allergic" subjects. No such correlation was found, though of course carbonic anhydrase (and probably its level in the gastric mucosa) is important in the secretion of gastric acid. Gaddum (1951) has pointed out that both histaminase may be reduced in allergic individuals and more histamine is released. The former finding may be important in indicating a biochemical difference in the allergic individual; the latter, unless related to a higher histamine content of organs, is more related to the reaction itself. The speed of acetylation of histamine in the liver may be significant in an overwhelming anaphylactic response as determining reduction of the impact of the reaction.

Now while antihistaminic substances have been rightly employed in therapy of the allergic state, since they act by inhibiting the action of histamine evidently by competition at the end organ responding to it, there is another field of approach. It may be that in view of the lack of knowledge concerning the condition allowing the development of the allergic state, the emphasis on inhibition of the activity of released substances has perhaps allowed neglect of therapy to inhibit the anaphylactic process. It is well known that salicylate compounds (Smith, 1949) are of value in the treatment of recognized allergic conditions—for example, rheumatic fever. It was also shown by Coburn and Kapp (1943) some ten years ago that

FIGURE II.[1]

Responses of the guinea-pig jejunum to perfusate and histamine in sensitized guinea-pig lung experiments with Tyrode solution as the perfusing fluid. At H, response to 0·1 microgramme of histamine, at h, to 0·04 microgramme, at h′ to 0·02 microgramme, at h″ to 0·01 microgramme, and the unlettered contractions are reactions to 0·1 microgramme of histamine. Upper panel: no salicylate in perfusion fluid; at A, failure of response to control perfusate obtained prior to injection of antigen. At B and C, response to perfusate containing histamine and SRS. after injection of antigen (0 to 5 and 5 to 10 minutes after injection respectively). Lower panel: salicylate perfusion; at B and C, no response to perfusate obtained after intraarterial injection of antigen, and lack of response to control perfusate obtained to injection at A.

salicylate would prevent precipitate formation between egg albumen and antibody. Jager and Nickerson (1947) have recently shown that salicylate prevents antibody formation to H and O antigen in rheumatic fever patients injected with typhoid vaccines. On the other hand, Perry (1941) earlier was unable to affect the development of agglutinins following TAB vaccination in the presence of acetyl salicylic acid. The explanation of this failure will appear

[1] The experiments shown in Figures II and III are recorded in the paper by Trethewie (1951).

clear in a moment. I have been able to show (Trethewie, 1951*a*) that sodium salicylate and acetyl salicylic acid inhibit the release of histamine and S.R.S. from the perfused sensitized guinea-pig lung when it is exposed to antigen. This is illustrated in Figures II and III. The release of S.R.S. is not inhibited quite so completely by lower levels of salicylate as is histamine. The histamine release is completely inhibited only when the concentration of sodium salicylate is somewhere between 25 and 50 milligrammes per 100 millilitres of fluid. Acetyl salicylic acid had similar activity. Now the child suffering from rheumatic fever is not rendered afebrile by salicylate unless the blood concentration is 30 to 35 milligrammes per 100 millilitres (Peters, 1947). This close agreement in effectiveness in anaphylaxis and in rheumatic fever is probably more than fortuitous. The conclusion is likely that either histamine or some other substance released in anaphylaxis is responsible for the pyrexia. Since phagocytosis is not a well developed function of the Aschoff cell and the Aschoff node ultimately gives rise to the crippling cardiac fibrosis of rheumatic fever, and since the only known function of histamine in cellular reaction (other than vasodilator and permeability effects) is to stimulate reticulo-endothelial cells to phagocytic activity, it could be concluded that in the absence of any other effect of salicylate such therapy would have no effect on the fibrosis. Such would seem to be the case. Many pædiatricians hold to the opinion that salicylate therapy has no effect on the fibrosis. The failure of Perry to inhibit agglutinin production while Jager and Nickerson were successful could well be due to the quantitative element we have demonstrated. However, the inhibition of antibody production is a different matter from inhibition of antigen-antibody reaction. The findings suggest that both the production of antibodies and the antigen-antibody reaction are inhibited. It may be that salicylate compounds block the antibody receptor surface of the antibody obtained from the lymphocyte, which would account for a proportionate effect in relation to concentration as I have shown, though admittedly the concentration for complete inhibition is high. This is illustrated in Figure I. This would, nevertheless, also account for suppression of production of antibody by the cell surface in sensitization if the precursor structure of the cell lipoprotein which would form antibody was blocked by salicylate. In this way equally the antigen-antibody reaction would be inhibited, as the linkages to take part in the reaction would be blocked. This work is of interest to allergists, as it is to be remembered that salicylate forms a part of one standard mixture used in the treatment of asthma. Many physicians have had experience of salicylate compounds in large doses interrupting the distressing complaint of *status asthmaticus* when other therapy has failed, though admittedly it may not be effective. On the other hand, it is to be remembered that Hungarian workers (Keleman *et alii*, 1950) consider that the effect of salicylate is through a pituitary adrenal mechanism; but I have shown it active in the absence of the pituitary and adrenal.

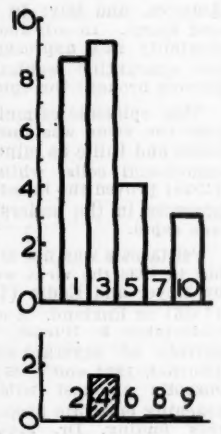

FIGURE III.[1]

Comparison of output of histamine from sensitized perfused lung with and without added sodium salicylate (100 milligrammes per 100 millilitres) in the perfusion fluid. Ordinates: output expressed as 1000 x milligrammes of histamine liberated per milligramme of albumin injected per microgramme histamine content of lung per gramme. Abscissæ: Animal Numbers 1, 3, 5, 7, 10: control pigs (open columns); 2, 4, 6, 8, 9: salicylate-perfused pigs (cross-hatched). In Experiment 4 (salicylate), the salicylate was greatly reduced at the time of injection of antigen by injecting salicylate-free Tyrode solution intra-arterially at the same time.

Now another feature of this phenomenon has been investigated. Independently by Castex (1950) in the Argentine, by Reimann and co-workers (1951) in Turkey and by myself (Trethewie, 1951*b*) here in Australia, the proposition has been put forward that the value of para-amino salicylate therapy in tuberculosis is due to its anti-allergic effect. All workers point out that para-amino salicylate is of particular value in the exudative or "allergic" form of tuberculosis. Castex concluded from careful clinical observation that it was probably an anti-allergic effect of para-amino salicylate that was valuable when taken in conjunction with the value of other salicylate compounds in the allergic condition. Reimann *et alii* (1951) also reached the same conclusion on similar grounds, pointing out that even with favourable therapeutic response patients treated with PAS alone commonly showed little bacterial sputum change, and he pointed out that PAS inhibited anaphylaxis in the intact rabbit with blood levels corresponding to therapeutic levels in tuberculosis. The significance of the allergic aspect in the type of patient responding to PAS and the anti-allergic effect of salicylate compounds, coupled with quantitative studies of the level of PAS in the perfusion fluid inhibiting anaphylaxis in the isolated perfused lung with inhibition of release of histamine and S.R.S., indicated to me that its value here was partly its anti-allergic effect. I have also pointed out that much evidence indicates that antihistamines reduce the tuberculin response (Trethewie, 1952). Of further interest is the reported finding of Matteucci and Vernacchia (1950) that histamine stimulates the growth of tubercle cultures, and that in guinea-pigs the tuberculous process is rendered more severe by histamine and is attenuated by antihistaminics. However, Dr. Gardner, Dr. Cowling and I (Trethewie, Gardner and Cowling, 1952) were unable to confirm these findings for tubercle cultures, though it is interesting to note that one of the first successful media for their culture was basically potato, which is rich in histamine.

Thus the significance of allergy in other diseases not primarily allergic indicates that when one is considering fundamental phenomena the application in practice may often be a broad one and have further significance.

In conclusion, it may be pointed out that many of the fundamental phenomena in anaphylaxis and presumably in allergy are known, but their relative importances are not yet fully assessed and the fundamental cause determining why one person can readily become allergic at present largely eludes us.

[1] The experiments shown in Figures II and III are recorded in the paper by Trethewie (1951*a*).

References.

ANREP, G. V., AYADI, M. S., BARSOUM, G. S., SMITH, J. R., and TALAAT, M. M. (1944), "The Excretion of Histamine in the Urine", *J. Physiol.*, 103: 155.

BARTOSCH, R., FELDBERG, W., and NAGEL, E. (1932), *"Das Freiwerden eines histaminähnlichen Stoffes bei der Anaphylaxie des Meerschweinchens"*, *Arch. f. d. ges. Physiol.*, 230: 129.

BLASCHKO, H. (1950), private communication.

CASTEX, M. R. (1950), *"La sal sodica del ácido paraaminosalicílico es un antialérgico efectivo"*, *Prensa méd. argent.*, 37: 639.

COBURN, A. F., and CAPP, E. M. (1943), "Effect of Salicylates on Precipitation of Antigen with Antibody", *J. Exper. Med.*, 77: 173.

DRAGSTEDT, C. A., and GEBAUER-FUELNEGG, E. (1932), *"Studies* in Anaphylaxis; Appearance of Physiologically Active Substance during Anaphylactic Shock", *Am. J. Physiol.*, 102: *512.*

FELDBERG, W., and O'CONNOR, W. J. (1937), "The Liberation of Histamine from the Perfused Lung by Peptone", *J. Physiol., 90: 288.*

GADDUM, J. H. (1951), "The Metabolism of Histamine", *Brit. M. J.*, 2: 987.

JAGER, B. V., and NICKERSON, M. (1947), "Altered Response of Human Beings to Intramuscular Administration of Typhoid Vaccine During Massive Salicylate Therapy", *Am. J. Med.*, 3: 408.

JAQUES, L. B., and WATERS, E. T. *(1941), "The Identity and* Origin of the Anticoagulant of Anaphylactic Shock in the Dog", *J. Physiol.*, 99: 454.

Keleman, E., Majoras, M., Iványi, J., and Kovács, K. (1950), "Salicylates, Stress and Cortisone", *Experientia*, 6: 435.
Kellaway, C. H. (1947), "Perfusion Experiment in Study of Tissue Injury", *Edinburgh M. J.*, 54: 333.
Kellaway, C. H., and Trethewie, E. R. (1939), unpublished experiments.
Kellaway, C. H., and Trethewie, E. R. (1940), "The Liberation of a Slow-Reacting Smooth Muscle-Stimulating Substance in Anaphylaxis", *Quart. J. Exper. Physiol.*, 30: 121.
Matteucci, L., and Vernacchia, A. (1950), "*Il rapporto istamina-ist-aminasi fattore determinante nella patogenesi dell'allergia tubercolare*", *Bull. sc. med.*, 122: 287.
Millican, R. C., Rosenthal, S. M., and Tabor, H. (1949), "On Metabolism of Histamine; Urinary Excretion following Oral Administration; Conjugation in Vitro", *J. Pharmacol. & Exper. Therap.*, 97: 4.
Perry, C. B. (1941), "Action of Salicylates on Development of Antibodies following Anti-Typhoid Inoculation", *J. Path. & Bact.*, 53: 291.
Peters, J. T. (1947), "The Necessity and Possibility of Giving Detoxified Large Oral Doses of Salicylates in the Treatment of Rheumatic Fever in Order to Prevent or cure the Inflammatory Stage of Carditis", *Acta med. scandinav.*, 128: 66.
Reimann, F., Laqueur, W., and Ulagay, I. (1951), "The Anti-Allergic Properties of Para-amino-salicylic Acid (P.A.S.)", *Istambul Contrib. Clin. Sc.*, 1: 171.
Rocha e Silva, M. (1952), "Concerning the Mechanism of Anaphylaxis and Allergy", *Brit. M. J.*, 1: 779.
Smith, P. K. (1949), "Certain Aspects of the Pharmacology of the Salicylates", *Pharmacol. Rev.*, 1: 353.
Tabor, H., and Mossetig, E. (1949), unpublished work quoted by Millican, R. C., Rosenthal, S. M., and Tabor, H. (1949), *loco citato*.
Trethewie, E. R. (1938), unpublished experiments.
Trethewie, E. R. (1939), "Comparison of Hæmolysis and Liberation of Histamine by Two Australian Snake Venoms", *Australian J. Exper. Biol. & Med. Sc.*, 17: 145.
Trethewie, E. R. (1945*a*), "Anticoagulant Activity of Diluted Blood Perfused Through the Lung", *Australian J. Exper. Biol. & Med. Sc.*, 23: 231.
Trethewie, E. R. (1945*b*), "Anticoagulant Effect of Perfusing Organs: Liver, Kidney and Lung", M. J. Australia, 2: 328.
Trethewie, E. R. (1947), "Mustard Hypersensitivity and Achlorhydria", M. J. Australia, 1: 633.
Trethewie, E. R. (1949), "The Role of Histamine in 'Non-Specific' Immunity", M. J. Australia, 1: 597.
Trethewie, E. R. (1951*a*), "The Influence of Sodium Salicylate and Acetyl Salicylic Acid on the Release of Histamine in Anaphylaxis", *Australian J. Exper. Biol. & Med. Sc.*, 29: 443.
Trethewie, E. R. (1951*b*), M. J. Australia, 2: 718.
Trethewie, E. R. (1952*a*), M. J. Australia, 2: 526.
Trethewie, E. R. (1952*b*), "The Effect of Para-amino-salicylic Acid on the Allergic Process", M. J. Australia, 1: 638.
Trethewie, E. R., and Day, A. J. (1949*a*), "Tissue Injury by Australian Black Snake Venom and 'Bound' Histamine", *Australian J. Exper. Biol & Med. Sc.*, 27: 385.
Trethewie, E. R., and Day, A. J. (1949*b*), "Ease of Release of Histamine from the Lungs of Guinea Pigs", *Australian J. Exper. Biol. & Med. Sc.*, 27: 417.
Trethewie, E. R., and Day, A. J. (1949*c*), "Some Observations on the Secretion of Acid", *Australian J. Exper. Biol. & Med. Sc.*, 27: 429.
Trethewie, E. R., and Gaffney, F. M. (1949), "The Influence of Serum Gonadotropic Hormone on the Histamine Content of and Ease of Release of Histamine by Trypsin from the Guinea-Pig Lung", *Australian J. Exper. Biol. & Med. Sc.*, 27: 523.
Trethewie, E. R., and Gaffney, F. M. (1950), "The Influence of Gonadal Hormones on the Histamine Content of the Lungs of Guinea-Pigs", *Australian J. Exper. Biol. & Med. Sc.*, 28: 589.
Trethewie, E. R., and Gaffney, F. M. (1951), "The Presence of 'Bound' Histamine following the Injection of Histamine Acid-Phosphate into Perfused Liver", *Australian J. Exper. Biol. & Med. Sc.*, 29: 67.
Trethewie, E. R., Gardner, H. J., and Cowling, D. C. (1952), unpublished experiments.
Ungar, G., and Mist, S. K. (1949), "Observations on the Release of Serum Fibrinolysin by Specific Antigen, Peptone and Certain Polysaccharides", *J. Exper. Med.*, 90: 39.

PSITTACOSIS IN AUSTRALIA.

By E. L. French,[1] R. A. Joske,[1] D. H. Bodycomb, E. F. Mackenzie and W. R. Rigg.

From the Department of Experimental Medicine of the University of Melbourne, the Clinical Research Unit of the Walter and Eliza Hall Institute of Medical Research and the Royal Melbourne Hospital, and the Department of Health, Victoria.

Psittacosis was first recognized by Juergensen in 1876, and three years later the first definite epidemic occurred in Ulster, Switzerland, when seven cases of "pneumo-typhus", with three deaths, occurred in the household of a bird fancier following the reception from Hamburg of a collection of birds, some of which became ill and died. In 1895 the term "psittacosis" was first used by Morange; but although further scattered cases and minor epidemics occurred from time to time, psittacosis received little scientific study and the ætiology and natural history of the disease remained obscure.

In 1929 and 1930, at about the time when present methods of study of viruses matured, psittacosis became a matter of acute medical interest and suddenly reached epidemic proportions. The outbreak began in Argentina early in 1929, following the delivery from Brazil of a consignment of 5000 birds. It quickly spread to England and North America, and later to many European countries, Algeria and Egypt. In all, about 800 cases were reported, with a mortality rate approaching 20%, before stringent import and quarantine regulations on the transport and sale of parrots brought the epidemic under control (Meyer, 1942).

This epidemic stimulated study of the disease, and in 1930 the virus was independently described by Levinthal, Coles and Lillie as minute spherical bodies within reticulo-endothelial cells, whilst Bedson, Western and Simpson (1930) proved the infective agent to be a virus. Subsequent progress in the understanding of both virus and disease was rapid.

Psittacosis was not at first thought to occur in Australia, but in 1934 the virus was isolated from Australian parrots by Meyer and Eddie (1934) in America and by Levinthal (1935) in England. A survey of native birds was therefore undertaken by Burnet, who isolated psittacosis virus from parrots of several species in south-eastern Australia (Burnet, 1934 and 1935). He discovered the disease to be enzootic amongst wild and captive parrots, at times reaching epizootic proportions (Burnet, 1939*a*). Because of this finding, Dr. J. H. L. Cumpston, Commonwealth Director-General of Health at that time, appealed for a greater awareness of the disease (Cumpston, 1935), and for information to enable a "full epidemiological survey" to be made ("Current Comment", 1934). The response was enthusiastic, and in 1936 the then Chief Health Officer for the State of Victoria reported as follows (Robertson, 1936):

> The request was too copiously complied with—so much so that it was impracticable to bacteriologically investigate all notifications.
>
> Many cases in humans were reported which, under normal circumstances, would have been regarded as influenza and which were in fact the condition usually diagnosed as influenza.

Nevertheless the first human cases soon came to light. Love (1934) recorded a case from which Merrillees (1934) obtained an organism which was probably the psittacosis virus, although definitive proof is lacking. Then Ross and Rabinov (1936) described a minor epidemic in a Melbourne suburb, and Kennedy (1936) a case from a Victorian country district. The virus was isolated on both occasions (Burnet and Macnamara, 1936). Tremain (1938) isolated the virus from a dead Bengalese finch imported from China.

By 1936 eighteen cases had been investigated in Victoria, and in five the virus was isolated. All subjects had contact with birds of the parrot family.

Further investigation was proposed and methods of diagnosis and control were considered (Keogh, 1936). However, with the outbreak of the Second World War little further work was done on the problem of psittacosis in this country. No cases were notified for the years 1940 to 1944 inclusive. In 1945 six cases with two deaths were reported from Adelaide by Yeatman and McEwin (1945), and a further case was shown at a meeting of the South Australian Branch of the British Medical Association in 1952 (Southcott, 1952). In 1945 de Burgh *et alii* (1945) isolated a virus indistinguishable from that of psittacosis from a naturally occurring infection in mice.

In 1950 the National Health and Medical Research Council recommended that the name "ornithosis", first proposed by Meyer (1942), should be used for the disease, and that it should be notifiable in all States. This recommendation was adopted.

[1] Working with the aid of grant from the National Health and Medical Research Council of Australia.

The incidence of psittacosis and ornithosis in Australia is shown in Table I. The number of cases is remarkably few and it is possible that awareness of psittacosis is not well developed among Australian practitioners. The solitary notification from New South Wales is especially striking. Psittacosis virus has been found in parrots from all States of the Commonwealth (Burnet, 1939b).

TABLE I.

The Incidence of Psittacosis. Number of Cases of Psittacosis Reported in Australia for the Years 1936 to 1953.

Year.	Victoria.	South Australia.	New South Wales.
1936	7	—	—
1937	—	—	—
1938	2	—	—
1939	2	—	—
1940	—	—	—
1941	—	—	—
1942	—	—	—
1943	—	—	—
1944	—	—	—
1945	—	2	—
1946	—	—	—
1947	2	—	—
1948	—	—	—
1949	—	2	—
1950	—	1	—
1951	—	—	—
1952	1	—	—
1953	4	—	1

The present paper reports further recent cases of ornithosis and discusses briefly the epidemiology, diagnosis and treatment of the disease.

Reports of Cases.

CASE I.—S., a male truck driver, aged forty-six years, was first examined at his home on September 17, 1952, when he complained of malaise, headache, sore throat, constipation and low back pain.

On examination of the patient, his temperature was 100° F., his pulse rate was 90 per minute, and his respiration rate was 18 per minute. He had moderately severe acute pharyngitis, and treatment with "Sulphatriad" was begun.

Two days later his condition had deteriorated, his temperature was 102° F., and he had developed a cough with rusty sputum. His previous symptoms persisted, and there were dullness and crepitations at the base of his right lung. A provisional diagnosis was made of "virus pneumonia". "Sulphatriad" therapy was discontinued and "Distaquaine" penicillin, 900,000 units, was given on this and the five succeeding days.

His general condition began to improve, although some frank blood was present in his sputum. He made a final complete recovery, and no further antibiotic therapy was necessary.

When first examined the patient had stated that he kept parrots in an aviary as a hobby, but he insisted that all of them were healthy. However, three days later, on September 20, he admitted that one of the birds had died that morning. The diagnosis of psittacosis was made and specimens of blood and sputum were taken for examination (*vide infra*). The virus of psittacosis was found to be present in the sputum, but the blood gave negative results to complement fixation tests for psittacosis until on repeated testing a positive titre of one in five was found on March 17, 1953, six months after his illness.

CASE II.—M., a school-teacher, aged fifty-five years, was admitted to hospital on December 5, 1952. For five days he had suffered from increasing tiredness and anorexia, and on the day of his admission he developed aching central abdominal pain and severe persistent substernal pain. There was no history of cough, pharyngitis or skin rash. He was a keeper of budgerigars, but had not noted any illness in the birds before the onset of his own illness.

On examination, the patient was flushed, with a temperature of 102° F., a pulse rate of 76 per minute, and a respiration rate of 20 per minute. There was slight neck stiffness but no other abnormal physical sign.

Investigations gave the following information. The hæmoglobin value was 14·9 grammes per 100 cubic centimetres, the white cell count was 10,000 per cubic millimetre, slight lymphocytosis being present. Agglutination tests with *Salmonella typhi* "H" and "O", *S. paratyphi A and B*, *Brucella abortus* and *Proteus* OX19 all gave negative results on two occasions. Repeated attempts at blood culture produced no growth. The cerebro-spinal fluid was normal in appearance, with no pleocytosis, and normal protein, sugar and chloride contents. An X-ray examination of the chest gave normal findings at the time of the patient's admission to hospital; but a second film one week later showed a vague opaque area in the left hilar region, probably in the apex of the lower lobe of the left lung. A complement fixation test with psittacosis antigen gave a positive result with steadily rising titres of one in five on December 15, 1952, one in 10 on January 7, 1953, one in 20 on January 12, and one in 80 on February 3. Therapy began on December 10, 1952, with the oral administration of chloramphenicol, two grammes being given daily in divided doses. There was a prompt subsidence of pyrexia (Figure I) and an improvement in the patient's sense of well-being. Six days later (December 15) the dose of chloramphenicol was halved, and therapy ceased next day. He was discharged from hospital on December 16.

The patient was readmitted to hospital for review on January 6, 1953. He had remained well, with no cough or rigors. Clinical and radiological examination of his chest revealed no abnormality. He had a slight pyrexia of 99° F. which subsided uneventfully (Figure I).

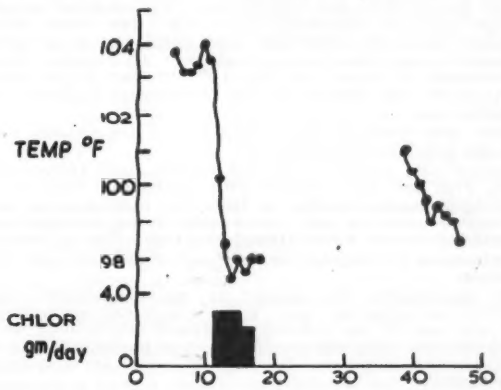

FIGURE I.

Temperature chart from a case of psittacosis (Case II), showing response to chloramphenicol. Abscissa—day of disease.

CASE III.—A., a male veterinary scientist, aged fifty-six years, was first examined at his home on April 17, 1953. Two days previously he had developed vague left-sided chest pain followed by fever, nausea and headache. Two weeks previously he had had contact with some birds which had died of an unspecified illness. He did not examine them closely, but received them in the same package as a dead seal upon which he had conducted an extensive post-mortem examination.

On examination, the patient was pale and looked ill. His temperature was 101·2° F., his respiration rate was increased, and there was a small area of consolidation with fine crepitations at the base of the right lung. It was considered that he was suffering from influenza with early bronchopneumonia, and he was treated with "sulphadiazine" and "aspirin". His condition did not improve, and 400,000 units of penicillin were given daily by intramuscular injection until April 21. The chest signs cleared, but headache, nausea, anorexia and sleeplessness persisted.

On April 21 he had a severe rigor lasting ten minutes and intensive headache. A blood culture was attempted, but no growth resulted, and blood examination revealed a leucocyte count of 11,300 per cubic millimetre with a polymorphonuclear leucocytosis. He was admitted to hospital for further investigation.

When the patient was examined after his admission to hospital his temperature was 99·4° F., his pulse rate 80 per minute, and his respiration rate 20 per minute. No abnormal physical signs were elicited. The spleen was not palpable.

He was extensively investigated. His hæmoglobin value was 17·1 grammes per 100 cubic centimetres, and the white cell count was 8000 per cubic millimetre with slight monocytosis. The Mantoux test produced a positive reaction to

0·1 cubic centimetre of one in 1000 old tuberculin, the control injection giving a negative result, and further attempts at blood culture produced no growth; agglutination tests with *S. typhi* "H" and "O", *S. paratyphi* "A" and "B", *Br. abortus* and *Proteus* OXK and OX19 gave negative results. No cold agglutinins were present in his serum, the Paul-Bunnell test produced a negative result, no pathogenic organisms were isolated from his sputum, urine and fæces, and an X-ray film of his chest was reported as being within normal limits. The complement fixation titre for psittacosis was less than one in five on April 22, 1953. No therapy was given, and he was discharged from hospital on April 25.

He was examined again in his home on May 4. He had remained well until that day, when a feeling of malaise had caused him to return home from work. His temperature was 99° F., but no abnormal physical signs were found on examination of the patient. Over the succeeding five days he developed lassitude, nausea and severe headache, and his temperature was elevated to 103° F.

On May 10, a small patch of fine crepitations was detected over the lower lobe of the right lung, and penicillin, 1,200,000 units daily, was again administered, but with no improvement. Next day (May 11), advice was received that the complement fixation test for psittacosis had produced a positive result at a dilution of one in seven, and therapy with chloramphenicol was begun, later changed to terramycin (750 milligrammes being given orally every six hours), and he showed immediate improvement, his headache easing and his sense of well-being increasing. The chest signs gradually spread to affect both lung fields, but at no time was there cough, chest pain or sputum. All physical signs had reverted to normal on May 18. On May 25 the titre of one in 20 was obtained in the complement fixation test for psittacosis.

When last examined three months later the patient was well and free from all symptoms.

CASE IV.—F., a male laboratory worker, aged thirty-nine years, who had had contact with psittacosis virus, was admitted to hospital on June 4, 1953. For eight days he had had malaise, anorexia and a dry cough without sputum, and for two days steady severe frontal headache. The day before his admission to hospital he developed rigors, shivers and sweating.

On examination, he looked ill; his temperature was 101·2° F., his pulse rate was 78 per minute and his respiration rate was 14 per minute. Scattered crepitations were heard over the right mid-zone of his chest posteriorly, and at the base of the left lung. No other abnormal physical signs were elicited.

Investigations revealed that the hæmoglobin value was 15·8 grammes per 100 cubic centimetres, the white cell count was 10,000 per cubic millimetre, and monocytosis was present. Agglutination tests gave negative results with *S. typhi* "O" and "H", *S. partyphi* "A" and "B", *Br. abortus*, *Proteus* OX19 and OXK. No cold agglutinins were demonstrable in the serum. Attempted blood culture produced no growth. X-ray examination of the chest revealed vague loss of translucency at the base of the left lung, and some "ground glass" opacity peripherally in the right lower mid-zone (Figure II). The complement fixation test with psittacosis antigen, which was known to have produced a negative result previously, produced a positive result at a dilution of one in 10 on July 28. The virus was not isolated from his sputum or blood.

Therapy began with aureomycin, 250 milligrammes being given every six hours orally. This produced some vomiting, so on June 6 the route of administration was changed to the intravenous one. Improvement was slow, and the patient developed pharyngitis and diarrhœa. Abnormal physical signs persisted in his chest, and bronchial breathing developed over the right mid-zone posteriorly. At no stage was enlargement of his spleen detected. His temperature became normal on June 14, and aureomycin therapy was stopped next day. He was discharged from hospital on June 18.

When the patient was last examined, on July 20, his temperature was normal, and his chest was normal on physical examination, although a little faint hazy opacity persisted in the radiological picture (Figure III).

CASES V AND VI.—On November 15, 1952, two weeks before the onset of symptoms, M. (Case II) had obtained three budgerigars from a breeder, G. One of these escaped from captivity before the beginning of M.'s illness. The other two were examined, but did not show evidence of psittacosis. Neither G. nor his sister Ju. gave any history suggestive of psittacosis; but both gave positive results to complement fixation tests, with titres of one in 10 (February 16, 1953) and one in 10 (April 16, 1953) respectively. Both had close contact with budgerigars.

In addition to these cases, a specimen of serum was received from New South Wales in May, 1953, which gave a positive result to the complement fixation test for psittacosis with a titre of one in 10.

Virological Investigations on the Present Series of Cases.

Isolation of Virus.

From Case I a specimen of sputum in a sterile jar was obtained on September 24, 1952. This was diluted with broth and inoculated intraperitoneally in 10 mice, aged four weeks. After nine days some of the mice had slightly ruffled fur and five of them were sacrificed. The abdomen was found to contain a mucinous exudate with some fibrinous exudate over the liver and spleen, the latter organ being enlarged to two or three times the normal size in each instance. Attempts at ordinary bacteriological cultures of the peritoneal exudate were unsuccessful, and mixed spleen and liver emulsions were passed to another group of mice intraperitoneally as well as to chick embryos five days old via the yolk sac. No antibiotic was used with any of these inoculations. After five days' incubation at 35° C. the chick embryos were observed to be dying, and impression smears of the yolk sac strained by Casteneda's and Macciavello's methods revealed the presence of the characteristic elementary bodies of the psittacosis-lymphogranuloma group of viruses. Suspensions of infected yolk sac were used to infect four weeks old mice peritoneally, and eight days old chick embryos suballantoically. The mice died in three days with typical peritoneal exudate and enlarged spleens and livers. The chick embryos died between the fifth and seventh days after inoculation, when the allantoic fluid was found to be gelatinous and opaque and containing enormous numbers of LCL bodies. This infected allantoic fluid was steamed and used as an antigen with a known positive psittacosis serum in a complement-fixation test. A positive result confirmed the identity of the isolated virus as a strain of psittacosis virus.

Complement-Fixation Test.

Psittacosis complement-fixing antigen was prepared by inoculating eight to nine days old chick embryos with the strain Blount (Burnet and Macnamara, 1936), and harvesting and steaming at 100° C. for twenty minutes the infected allantoic fluid harvested on the fourth to fifth day. The optimum dilution of antigen for use in the test was determined by performing a "chessboard" titration of the steamed antigen with a known "positive" specimen of serum. The highest dilution of antigen giving the highest titre with the serum was chosen for use in the test with unknown sera.

The complement was from a pool of male guinea-pig sera kept frozen at –20° C., and was titrated on the day of the test in the presence of the antigen and a normal allantoic fluid control. The 50% hæmolysis end point was used, and three times this amount of complement was used in the test proper.

In the test proper dilutions beginning at 1·5 of the patient's sera previously heated to 56° C. for thirty minutes or 62° C. for fifteen minutes were run against the fixed optimum dilution of antigen in the presence of $3HD_{50}$ of complement, fixation being allowed to proceed for thirty minutes at room temperature followed by sixty minutes at 37° C. in a water bath. The usual sensitized sheep red cell suspension was used as the indicator system. The end point was that dilution of serum showing 50% hæmolysis in the test, Serum without antigen and serum with normal allantoic fluid antigen were run as controls. The complement dilution used was check-titrated with each test. All dilutions were made in calcium magnesium saline.[1]

[1] Calcium magnesium saline was modified from the formula published by Mayer, Osler, Bier and Heidelberger (1946) as follows: Sodium chloride 8·5 grammes, anhydrous calcium chloride 0·0275 gramme, magnesium chloride (crystalline) 0·192 gramme, boric acid (crystalline) 1·203 grammes, sodium borate (crystalline) 0·052 gramme, in distilled water 1·0 litre.

Discussion.

Psittacosis is a disease of wild birds, and is enzootic among wild birds in many parts of the world (Meyer and Eddie, 1939). Young birds in the nest are probably infected from their infected but asymptomatic parents. A few of the nestlings die, but the majority survive. The survivors appear well but remain carriers of the disease for up to a year, harbouring the virus in the spleen, which is enlarged. Occasionally the balance between bird and virus is disturbed. An epizootic of psittacosis occurs, and large numbers of birds die (Burnet, 1939*a*).

The disease affects many species of birds. At first it was considered to be confined to the parrot family (Order Psittaciformes), and Meyer (1942) recognized 33 species from 19 genera known to exhibit infection with the psittacosis virus. Australian species listed include the budgerigar, rosella, lorikeet, galah, grass parrot, ringneck-parrot, Port Lincoln parrot, Regent parrot, and King parrot (Burnet, 1935 and 1939*b*; Meyer and Eddie, 1939; Meyer, 1942).

Recent work has steadily widened this view of the natural ecology of psittacosis. In 1939 Haagen and Mauer showed the fulmar petrel to be a carrier of psittacosis in the Faroe Islands, and later work has greatly increased the known range of natural hosts of the virus. The list now includes, besides many species of parrot, finches, canaries, doves, pigeons, ducks, seagulls and domestic fowl. In Australia, finches (Tremain, 1938) and semi-wild pigeons from the cities (Burnet, 1953) have been shown to carry the virus. These non-psittacine species are able to transmit infection to man (Meyer, Eddie and Yanamura, 1942).

This wide distribution of the disease led Meyer (1942) to propose that the name "ornithosis" should be substituted for the old name "psittacosis". The National Health and Medical Research Council of Australia adopted this proposal in 1950.

The natural history of psittacosis in caged birds differs slightly from the picture seen in wild birds. The disease may be enzootic amongst birds kept in aviaries, as Meyer (1942) found in California, where breeders' aviaries were almost invariably infected. In these circumstances there is little obvious disease of the birds, but if they are kept in crowded, unhygienic conditions, an epizootic may result (Burnet and Macnamara, 1936). The virus may be introduced into a "clean" aviary by recently captured wild birds, as may occur when nestling parrots are captured in the bush and sent to city dealers for resale to the public. Epizootics with a high mortality have followed the addition of wild birds to collections of parrots in zoological gardens. On the other hand, professional budgerigar breeders rarely introduce wild birds to their collections.

Human infection from the extensive reservoir in wild and caged birds may result from direct contact—for example, feeding birds from the mouth, or "kissing"—or from inhalation of dust derived from droppings or infected birds. Laboratory infections are not uncommon, and the psittacosis virus is generally recognized as dangerous to handle in the laboratory. Direct human-to-human spread, presumably by droplet infection of the respiratory tract, is rare, although proven cases are on record.

Clinical Features and Diagnosis.

The classical clinical picture due to ornithosis is "atypical" or "virus" pneumonia.

After contact with infected birds (which may be slight), there is an incubation period of one to two weeks followed by the onset of malaise, fever, anorexia, vomiting, constipation and aching pains in the muscles. Rigors, sweats, epistaxis and pleural pain are not uncommon. Cough is usual, but sputum is absent or scanty, occasionally blood-stained. The pulse and respiratory rates are only slightly elevated.

Irregularly scattered patches of consolidation appear after three or four days. At this stage there may also be diarrhœa, skin rashes and splenomegaly. After another week gradual recovery and retrogression of abnormal physical signs take place, but convalescence is slow and relapses are common in the absence of specific therapy.

Occasional atypical cases are seen in which pulmonary involvement is absent, and present as "pyrexia of unknown origin". Subclinical infections are also known.

Laboratory investigations show the white cell count to be normal or slightly raised, cold agglutinins are not present in the serum, and agglutination with streptococcus MG strains is absent. The radiological appearances vary. The usual picture is of scattered areas of "ground-glass" consolidation in both lungs, without definite relation to broncho-pulmonary segments.

This picture is in no way specific to ornithosis, and is seen also in true (viral) influenza, "Q" fever, some infections with the virus of lymphocytic choriomeningitis, and "primary atypical (virus) pneumonia of unknown ætiology" (Finland and Dingle, 1942; James, 1953).

The diagnosis of psittacosis as the cause of atypical pneumonia depends upon four things: recognition of the clinical picture, establishment of contact of the patient with a source of infection, and in the laboratory isolation of the virus from the sputum and the obtaining of a positive response to the complement-fixation test for the virus of psittacosis. Specimens of sputum and blood should therefore be sent for laboratory examination, if possible before the institution of treatment.[1] A second specimen of blood should be procured two or three weeks later, so that any increase in antibody titre can be detected. A rising antibody titre is of much greater diagnostic value than a single estimation. In some cases (Case I) the production of antibody may be delayed for some months after infection.

The proportion of cases of "atypical pneumonia" due to ornithosis is unknown. In London, Crofton *et alii* (1951) studied a series of 110 patients admitted to hospital with pneumonia in one year and found 26 of them to show serological evidence of recent viral or rickettsial infection. Three had high titres of complement-fixing antibody to the psittacosis-lymphogranuloma group of viruses.

No detailed surveys of this kind have been made in Australia. No information about the ætiological agents in pneumonia in this country is available, and the frequency of virus and rickettsial pneumonia, especially ornithosis, is unknown. Now that specific chemotherapy is available, further epidemiological, clinical and laboratory study of the pneumonias in Australian practice is urgently needed.

The mortality rate of ornithosis approached 20% before the introduction of chemotherapy, but more recently has dropped to below 3%. Almost all deaths occur in patients aged over forty years or with extensive lung involvement. There is some evidence that in general infection is more serious if it is derived from parrots than from other birds.

Treatment.

The viruses of the psittacosis-lymphogranuloma group are unusual in being susceptible to chemotherapy. A large literature has accumulated about this and has been reviewed by Meyer (1952). The virus of psittacosis is unaffected by sulphonamides or streptomycin, both in man and in experimental animals. Penicillin in large doses inhibits the growth of the virus, and is efficacious in man, in mice and in some species of birds. However, there is evidence that suggests that while penicillin has an apparent curative effect, the host may become an immune carrier of the virus, so that the newer "broad-spectrum" antibiotics are now preferred in psittacosis.

The most efficient treatment is with aureomycin or terramycin. Chloramphenicol is both less effective and more toxic. Treatment with antibiotics such as aureomycin or terramycin in the early stages of the disease may be so rapidly efficacious as to delay or prevent any antibody

[1] Facilities for laboratory diagnosis of psittacosis are available at the Institute of Medical and Veterinary Science, Adelaide. The Commonwealth Serum Laboratories, Parkville, Victoria, hope to begin complement fixation tests for psittacosis in the near future.

response to infection, and serological tests may then give negative results for some time after the illness.

Patients with psittacosis should be regarded as infectious.

Control of the Disease.

Because of the vast reservoir of infected wild birds, eradication of psittacosis from Australia is impracticable and public health measures are restricted to control of birds known to be infected. The disease is notifiable in all States, but the paucity of reported cases suggests that the disease is either unrecognized or not reported to health authorities.

The danger is greatest in young, recently captured, wild birds, or in sick birds. Transmission of infection to man from healthy adult bird carriers is not common. In all cases the danger is greatly increased by keeping birds in crowded, dark, dirty conditions. Obviously sick birds should be destroyed and their bodies and droppings disinfected or destroyed.

Breeders of and dealers in budgerigars and native parrots are specially liable to infection. Such persons should be aware of the possibility of infection and guard against it.

Summary.

1. The history of psittacosis in Australia is briefly outlined and six further cases are reported, from one of which the virus was isolated. In all the diagnosis was confirmed by serological investigation.

2. The psittacosis virus is endemic among many species of Australian parrots and other birds.

3. Human infection may occur by droplet infection or by direct contact with infected birds. It is usually manifest as "virus pneumonia", but sometimes evidence of pulmonary involvement is lacking. Occasionally infection is subclinical. The differential diagnosis of "virus pneumonia" is briefly discussed.

4. Psittacosis responds rapidly to antibiotic therapy with aureomycin, terramycin or chloramphenicol.

5. Measures for the control of the disease are outlined.

6. Awareness of psittacosis is not yet well developed in Australia, and the ætiological factors in the atypical pneumonias are largely unknown. Further work on these problems in Australian practice is urgently needed.

Acknowledgements.

Our thanks are due to those practitioners who referred cases to us for study, and to members of the honorary medical staff of the Royal Melbourne Hospital for permission to publish reports of patients under their care. The figures quoted in Table I were supplied by Dr. A. J. Metcalfe, Commonwealth Director-General of Health, and are published with his permission. The photographs were taken by Mr. R. Ingles, clinical photographer, Royal Melbourne Hospital.

References.

BEDSON, S. P., WESTERN, G. T., and SIMPSON, S. L. (1930), "Further Observations on the Ætiology of Psittacosis", *Lancet*, 1: 345.

DE BURGH, P., JACKSON, A. V., and WILLIAMS, S. E. (1945), "Spontaneous Infection of Laboratory Mice with a Psittacosis-like Organism", *Aust. J. Exper. Biol. & Med. Sc.*, 23: 107.

BURNET, F. M. (1934), "Psittacosis in Australian Parrots", M. J. AUSTRALIA, 2: 743.

BURNET, F. M. (1935), "Enzootic Psittacosis amongst Wild Australian Parrots", *J. Hyg.*, 35: 412.

BURNET, F. M. (1939a), "A Note on the Occurrence of Fatal Psittacosis in Parrots Living in the Wild State", M. J. AUSTRALIA, 1: 545.

BURNET, F. M. (1939b), "Psittacosis in Australia", "Proceedings Sixth Pacific Science Congress", 5: 349.

BURNET, F. M. (1953), "Natural History of Infectious Disease", Cambridge, 22.

BURNET, F. M., and MACNAMARA, J. (1936), "Human Psittacosis in Australia", M. J. AUSTRALIA, 2: 84.

CROFTON, J. W., FAWCETT, J. W., JAMES, D. G., SCADDING, J. G., MACRAE, A. D., and MARMION, B. P. (1951), "Pneumonia in West London, 1949-1950", *Brit. M. J.*, 2: 1368.

CUMPSTON, J. H. L. (1935), *"La psittacose en Australie", Bull. Office internat. d'hyg. pub.*, 27: 1362.

CURRENT COMMENT (1934), "Psittacosis in Australia", M. J. AUSTRALIA, 2: 791.

FINLAND, M., and DINGLE, J. H. (1942), "Virus Pneumonias. I. Pneumonias Associated with Known Non-bacterial Agents: Influenza, Psittacosis and Q. Fever", *New England J. Med.*, 227: 342.

JAMES, D. G. (1953), "Primary Atypical Pneumonia", *J.A.M.A.*, 151: 810.

KENNEDY, G. J. (1936), "Psittacosis in Man", M. J. AUSTRALIA, 1: 366.

KEOGH, E. V. (1936), "Report on Psittacosis", Report of the Federal Health Council of Australia, Ninth Session, Appendix III, Canberra, 23.

LEVINTHAL, W. (1935), "Recent Observations on Psittacosis", *Lancet*, 1: 1207.

LOVE, J. (1934), "Notes on a Suspected Case of Psittacosis", M. J. Australia, 2: 321.

MAYER, M. M., OSLER, A. G., BIER, O. G., and HEIDELBERGER, M. (1946), "The Activating Effect of Magnesium and other Cations on the Hæmolytic Function of Complement", *J. Exper. Med.*, 84: 535.

MERRILLEES, C. R. (1934), "Psittacosis in Australia", M. J. AUSTRALIA, 2: 320.

MEYER, K. F. (1942), "The Ecology of Psittacosis and Ornithosis", *Medicine*, 21: 175.

MEYER, K. F. (1952), "Psittacosis-Lymphogranuloma Group", in "Viral and Rickettsial Infections of Man", edited by T. M. Rivers, 2nd Edn., Lippincott, Philadelphia, Chapter 20: 440.

MEYER, K. F., and EDDIE, B. (1934), "Psittacosis in the Native Australian Budgerigars", *Proc. Soc. Exper. Biol. & Med.*, 31: 917.

MEYER, K. F., and EDDIE, B. (1939), "Psittacosis in Importations of Psittacine Birds from the South American and Australian Continent", *J. Infect. Dis.*, 65: 234.

MEYER, K. F., EDDIE, B., and YANAMURA, H. Y. (1942), "Ornithosis (Psittacosis) in Pigeons and its Relation to Human Pneumonitis", *Proc. Soc. Exper. Biol. & Med.*, 49: 609.

ROBERTSON, E. (1936), Report of the Federal Health Council of Australia, Ninth Session, Canberra, 17.

ROSS, F. C. H., and RABINOV, L. (1936), "A Local Epidemic of Human Psittacosis in Australia", M. J. AUSTRALIA, 1: 364.

SOUTHCOTT, R. V. (1952), Discussion on psittacosis, M. J. AUSTRALIA, 2: 644.

TREMAIN, A. R. (1938), "Some Aspects of Psittacosis and the Isolation of the Virus", M. J. AUSTRALIA, 2: 417.

YEATMAN, C., and MCEWIN, J. (1945), "Infections with Psittacosis in Adelaide", M. J. AUSTRALIA, 2: 109.

Reports of Cases.

A CASE OF TRACHEO-ŒSOPHAGEAL FISTULA.

By RICHARD FLYNN and JEANNE M. COLLISON,
Sydney.

C.J., A FEMALE BABY, was born on September 17, 1952. The mother had been well during her pregnancy. The fœtus was at term and was delivered by a demonstration instrumental delivery, after a second stage of labour lasting thirty-five minutes. The baby's birth weight was eight pounds and a quarter of an ounce, and her length 20·5 inches, and she seemed normal at birth.

The child made very feeble attempts to suck and was noticed to have a great deal of mucus in her mouth and pharynx, causing respiratory difficulty. Regurgitation of food by the nose was found on the third day after birth. The next day abdominal distension was noticed and much flatus was passed *per rectum*. After an unsuccessful attempt to tube feed the baby, an X-ray picture was taken after an "Abrodil" bolus had been given. The report was as follows:

> The swallow was obstructed about T2 level and the appearances suggest congenital atresia of the œsophagus [Figure I]. There appears to be a tracheo-œsophageal fistula about the level of the bifurcation of the trachea.

On September 23, the sixth day after birth, the child was taken to the operating theatre and under anæsthesia (cyclopropane, nitrous oxide and oxygen through an intratracheal catheter), given by Dr. W. I. T. Hotten, the chest was opened through a right-sided intercostal incision. The X-ray findings of œsophageal atresia and tracheo-œsophageal fistula were confirmed, and the tracheo-œsophageal fistula was closed. The proximal œsophageal pouch was isolated and an incision was made into it. A small urethral catheter was passed back up into the mouth, and the distal end of the œsophagus was pulled up. On account of the fragile nature of the œsophageal wall, which readily tore, it was difficult to insert the urethral catheter down into the stomach. Finally this was accomplished, and the two ends

of the œsophagus were united by interrupted silk sutures. The chest was closed around a De Pezzer catheter, and underwater drainage of the pleural cavity was set up.

After operation the catheter was removed from the œsophagus and the child was given nothing by mouth for five days. Fluid and electrolytes were given subcutaneously and 120 cubic centimetres of blood were given intravenously during this period. Antibiotics, ascorbic acid and oxygen (continuously administered) were used as supportive measures.

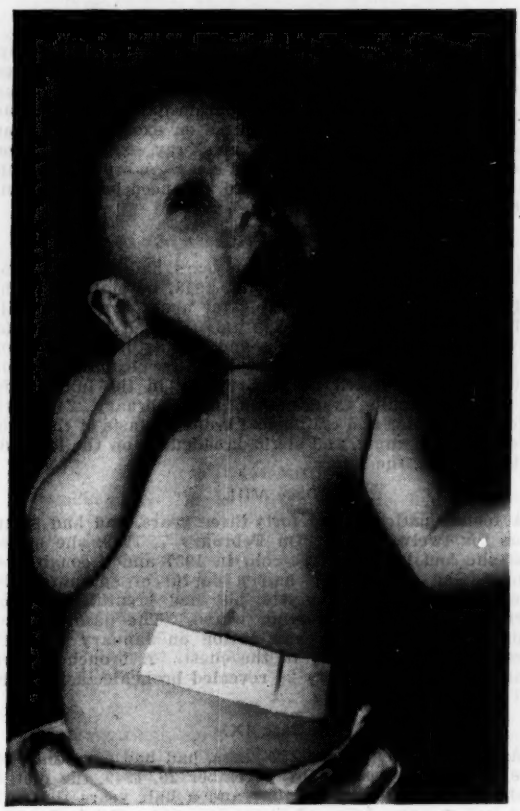

FIGURE III.

On the fifth day after operation a feeding of boiled water and indigo-carmine (sterile solution) was given orally. Blue staining was found almost immediately in the underwater drainage bottle, which indicated a breakdown in the anastomotic line. The child's condition was poor at this stage, the temperature having risen to 102° F. An X-ray film still showed some residual lung collapse but no evidence of consolidation in the lungs or fluid in the pleural cavity. The hæmoglobin value was 14·6 grammes *per centum* and the white cell count was 61,000 per cubic millimetre, 73% being neutrophile cells. In view of the finding at feeding, the child was again taken to the operating theatre and a gastrostomy was performed.

Feedings were then given by the gastrostomy tube. The antibiotics were changed and terramycin was given in the feedings. The baby was very ill for several days; the white blood cell count rose to 89,000 per cubic millimetre and toxic changes were found in the leucocytes. The temperature remained high (101° to 103° F.), but this gradually subsided over the next week. A further blood transfusion was given.

The child was fed on boiled water and indigo-carmine orally ten days after the gastrostomy was performed, and this feeding showed that a leak was still present. This was again done twenty days after gastrostomy, when a very small quantity seemed to have passed beyond the pleuro-œsophageal fistula.

A barium bolus was given, and this confirmed the fact that the œsophagus was patent and that there was a pleuro-œsophageal fistula. The gastrostomy tube was also found to be out of the stomach.

Oral feedings were commenced, and the child's weight, which was five pounds four ounces at this stage, gradually rose until on December 12 it was seven pounds five ounces, when the child was discharged from hospital.

Further barium studies showed that the fistula had closed and a pouch formed. The œsophagus had a very good lumen throughout, and the œsophageal pouch had become smaller at the last X-ray examination on April 9, 1953 (Figure II).

Since her discharge from hospital the baby has continued to gain in weight (Figure III, June 9, 1953).

Acknowledgements.

The late Dr. Norman Cunningham successfully met all the feeding problems which arose, and much of the credit for the successful outcome of the operation must be given to the skilful and devoted care of Sister F. Reynolds and Sister M. D. Frizell, of the staff of the King George V Memorial Hospital for Mothers and Babies.

BRONCHIECTASIS WITH NORMAL PLAIN X-RAY APPEARANCES.[1]

By H. B. HARWOOD,

Honorary Surgeon, Ear, Nose and Throat Department, Royal Prince Alfred Hospital, Sydney.

FROM time to time patients consult one with a history very suggestive of bronchiectasis or, at least, of very severe chronic bronchitis, but the plain chest X-ray film appearances seem to be within normal limits. Whilst the radiologist is usually ready to concede that it is possible that a bronchiectatic condition may even so be present but is perhaps masked by the shadow of the heart, this fact does not seem to be so readily recognized by some practitioners; otherwise we should not have the position of patients being told that there cannot be very much the matter as the plain X-ray film (or sometimes a number of plain X-ray films) has been reported on as being quite clear, thus indicating no definite lesion and hence no need for treatment.

Davidson (1948) points out that in suspected cases of bronchiectasis in which an ordinary X-ray film shows no definite evidence, the introduction of iodized oil will render obvious any dilatation of the small bronchial divisions.

For this paper I have collected 25 cases, 17 patients being adults of an average age of thirty-two years and eight being children. One of these was aged twelve years and all the others were under the age of nine years. All these patients had a history suggesting the possibility of bronchiectasis, but all had normal findings in the plain chest X-ray films as reported on by competent radiologists. Some of the adults had had several X-ray examinations extending over a number of years, all with the same report.

While the children may possibly escape correct diagnosis for several years, it does not seem right that adult patients can go on with definite bronchiectasis for eighteen years (the average in this series) before a diagnosis of bronchiectasis is made. When the story of the patient is listened to it seems remarkable today that the evidence that the plain X-ray film shows no abnormality should be acted on without any further examination such as bronchography or bronchoscopy.

The history of a bronchiectatic is usually fairly typical and there is no need to repeat it here, as it has been dis-

[1] Read at a meeting of the Oto-Laryngological Society of Australia (British Medical Association) on August 17, 1953.

cussed frequently in recent years. However, I most strongly suggest that when the typical history is given by the patient, no matter what the appearance of the plain skiagram may be, the history must be given full value, even if the chest examination as well as the skiagram does not reveal any definite signs of a lesion. Only if this is done and the patient is fully investigated by bronchoscopy and bronchography will a correct diagnosis be made, and, since most of these patients have a unilateral lesion, only by this investigation can they be given the chance of full recovery by surgical treatment, for which they are generally quite suitable if the diagnosis is made early.

In the list of patients below there are noted the date when they first consulted me and their age at that time, as well as the duration of their symptoms. There is also a brief history of their cough and its development, as well as a statement on the condition of their nasal sinuses. The date of the most recent plain skiagram is given, together with the radiologist's report. After this is given the date of the bronchographic examination with the findings, which the radiologist and I discuss together when he is reporting on them.

No mention is made of the bronchoscopic findings; but mucopus was present in varying amounts in all cases on lavage.

Case I.

A female patient, aged twenty-nine years, had had symptoms for twenty-six years. On May 20, 1946, she stated that she had had a cough since early childhood. She had had pneumonia ten years previously and pleurisy three or four times. She coughed up half a cupful of sputum daily. She had had no hæmoptysis. She had had an operation for pansinusitis. An X-ray examination on March 8, 1946, revealed no abnormality in her chest. A bronchographic examination on May 23 revealed some slight bronchiectasis at both lung bases.

Case II.

A female patient, aged twenty-one years, had had symptoms for twenty years. On February 10, 1947, she stated that at the age of nine months she had had measles and she had had a cough ever since. She coughed up a quarter of a cupful of sputum daily. She had had no hæmoptysis, but became breathless on exertion. Infection was present in both antra and in the left ethmoids. An X-ray examination on February 11 showed that the lung fields were clear. A bronchographic examination on May 22 revealed slight cylindrical bronchiectasis at the base of the left lung.

Case III.

A female patient, aged twenty-eight years, had had symptoms for twenty-five years. On June 2, 1947, she stated that she had had a cough for as long as she could remember. This followed whooping-cough, which she had had at the age of three years. She coughed up half a cupful of sputum daily. She had had an hæmoptysis two years earlier and she became breathless. Infection of the left antrum was present. An X-ray examination on May 15 showed the lung fields to appear clear. A bronchographic examination on June 12 revealed slight bronchiectasis at the base of the left lung and to a lesser degree at the base of the right lung.

Case IV.

A female patient, aged eighteen years, had had symptoms for eighteen years. On July 21, 1947, she stated that she had had pneumonia at the age of three months and had had a cough ever since. She had suffered from pneumonia as a child and three times since, the last attack having occurred two years earlier. She coughed up an eggcupful of sputum daily. She had had no hæmoptysis, but grew breathless and had had asthma. Infection was present in the right antrum. At an X-ray examination on June 30 the lung fields appeared clear. A bronchographic examination on July 24 revealed slight bronchiectatic changes in the posterior lobule of the lower lobe of the left lung.

Case V.

A female patient, aged thirty-three years, had had symptoms for twelve months. On June 29, 1948, she stated that she had developed a cough twelve months earlier. She had a moderate amount of sputum and had had two hæmoptyses five months and two weeks earlier respectively. The nasal sinuses appeared clear. At an X-ray examination on June 15 the lung fields appeared clear. A bronchographic examination on July 1 revealed cylindrical and saccular bronchiectasis, with atelectasis of the lower lobe of the left lung and a small area in the mid-zone of the right lung.

Case VI.

A male patient, aged twenty-nine years, had had symptoms for twenty-six years. On October 5, 1948, he stated that he had had a cough for as long as he could remember. He coughed up an eggcupful of sputum daily. He had had no hæmoptysis, but grew breathless on exertion. He had bilateral antral infection. At an X-ray examination on October 5 the lung fields appeared clear. A bronchographic examination on October 8 revealed advanced bronchiectasis at the left cardio-phrenic angle.

Case VII.

A male patient, aged thirty-nine years, had had symptoms for five years. On November 12, 1948, he stated that he had had a cough with wheezing for about five years. He had a moderate amount of sputum. He had had no hæmoptysis, but became breathless and had asthma. Infection of the right antrum was present. X-ray examination on November 19 revealed no abnormality in the lung fields. A bronchographic examination on February 24, 1949, revealed saccular and cylindrical bronchiectasis of the right cardio-phrenic angle, and slight broadening on the left side at the base of the lung.

Case VIII.

A female patient, aged forty-three years, had had symptoms for twelve years. On February 7, 1949, she stated that she had had a severe cold in 1937 and a cough ever since. She coughed up half a cupful of sputum daily. She had had no hæmoptysis, but had breathlessness on exertion, which was growing worse. The nasal sinuses appeared clear. X-ray examination on January 12, 1949, revealed no abnormality in the chest. A bronchographic examination on February 24 revealed bronchiectasis of the left cardio-phrenic angle.

Case IX.

A female patient, aged six years, had had symptoms for one and a half years. On November 29, 1949, the mother stated that at the age of four and a half years the child had had measles followed by pneumonia. She had had a cough ever since. She swallowed her sputum. Both antra were infected. An X-ray examination on June 10 revealed no abnormality in the lung fields. A bronchographic examination on December 8 revealed early cylindrical changes in the left anterior basal branches.

Case X.

A female patient, aged twelve years, had had symptoms for six years. On December 19, 1949, the mother stated that the child had had whooping-cough at the age of six years. About three and a half years earlier she herself had realized that the child had had a cough for quite a considerable time. The patient coughed up only a little sputum and had had no hæmoptysis. She had had nasal polypi removed and antral infection was present on both sides. A plain X-ray film taken on December 22 revealed no evidence of bronchiectasis. A bronchographic examination on January 26, 1950, revealed cylindrical bronchiectasis of the lower lobe of the left lung, particularly the basal branches. Slight suspicion was aroused about the anterior basal branches on the right side (Figure I).

Case XI.

A female patient, aged thirty-eight years, had had symptoms for thirty-seven years. On February 14, 1951, she stated that at the age of five months she had whooping-

cough and that she had had a cough ever since. She coughed up an eggcupful of sputum daily. Two years earlier she had streaks of blood in her sputum. She became breathless and had asthma. Infection of the right antrum was present. An X-ray examination on February 28 revealed no abnormality in the chest. A bronchographic examination on March 1 revealed slight dilatation of the bronchi at the left cardio-phrenic angle.

Case XII.

A female patient, aged three and a half years, had had symptoms for three years. On February 19, 1951, the mother stated that the child had had measles at the age of five months. She had had bronchitis at the age of twelve months and had had a cough ever since, periodically producing copious sputum. She had bilateral antral infection. At an X-ray examination on December 8 both lung fields appeared clear. A bronchographic examination on March 1, 1951, revealed slight bronchiectatic changes at the left cardio-phrenic angle.

Case XIII.

A male patient, aged seven years, had had symptoms for two and a half years. On May 11, 1951, the mother stated that he had had whooping-cough at the age of four and a half years. This was followed by bronchitis. He still had a moist cough, but not a great deal of sputum. He had had no hæmoptysis, but frequently suffered from colds. The nasal sinuses appeared clear. X-ray examination on May 14 revealed no evidence of bronchiectasis or any other abnormality of the lungs. A bronchographic examination on May 17 revealed advanced cylindrical bronchiectasis at the base of the left lung (Figure II).

Case XIV.

A female patient, aged thirty-one years, had had symptoms for twenty-five years. On June 29, 1951, she stated that she had had colds and bronchitis since early childhood. She coughed up a little sputum, occasionally slightly streaked with blood. The nasal sinuses appeared clear. At an X-ray examination on July 2 there was no evidence of any abnormality in the chest. A bronchographic examination on August 9 revealed an area in the lower lobe of the left lung at the cardio-phrenic angle which showed saccular dilatation.

Case XV.

A female patient, aged twenty-nine years, had had symptoms for five years. On February 13, 1952, she stated that she had had an hæmoptysis followed by a cough five years earlier. Two years later she coughed up a small piece of bone. She had another hæmoptysis two weeks prior to examination. She had had a little sputum for several years. The nasal sinuses appeared clear. At an X-ray examination on August 21 the chest was regarded as essentially normal. A bronchographic examination on February 28, 1952, revealed a localized bronchiectatic sac at the division of the lateral basal bronchus on the left side. No other lesion was seen.

Case XVI.

A female patient, aged forty-three years, had had symptoms for two years. On March 14, 1952, she stated that two years earlier she had had a severe cold in the chest. She had had a cough ever since whenever she lay on her back. She coughed up half a cupful of sputum daily. She had had no hæmoptysis. The nasal sinuses appeared clear. When radiologically examined on March 13, 1951, the chest had appeared clear. A later skiagram was also stated to show no abnormality, but was not obtainable. A bronchographic examination on March 20, 1952, revealed a localized area of cylindrical bronchiectasis in the middle lobe of the right lung (Figure III).

Case XVII.

A female patient, aged fifty-four years, had had symptoms for thirty-four years. On March 4, 1952, she stated that she had had a cough since she was a young adult. She coughed up half a cupful of sputum daily. She had had an hæmoptysis some years earlier and became breathless. She had undergone a double radical antrostomy five years earlier. Radiological examination on March 5 revealed no evidence of abnormality in the chest or mediastinum. Several previous X-ray examinations had been reported on in similar terms. A bronchographic examination on March 27 revealed crowding of the bronchi, and cylindrical bronchiectasis was present at the base of the left lung.

Case XVIII.

A female patient, aged thirty years, had had symptoms for twenty-six years. On March 21, 1952, she stated that she had had whooping-cough at the age of four years and had had a cough ever since. She had had pneumonia seven years prior to examination. She coughed up half a cupful of sputum daily. She had had no hæmoptysis, but suffered from breathlessness and wheezing. She had undergone a right radical antrostomy fifteen years earlier. An X-ray examination of the chest on March 27 revealed no abnormality. A bronchographic examination on April 3 revealed saccular bronchiectasis at the base of the left lung.

Case XIX.

A female patient, aged five years, had had symptoms for two and a half years. On May 16, 1952, the mother stated that after measles at the age of two and a half years the child had developed bronchopneumonia; she had then developed asthma and had had a cough ever since. She coughed up a moderate amount of sputum. The nasal sinuses appeared clear. A radiological examination on April 16 revealed no abnormality in the chest. A bronchographic examination on June 5 revealed early cylindrical bronchiectasis in the left basal bronchi; the right middle lobe bronchi also presented suspicious appearances (Figure IV).

Case XX.

A male patient, aged thirty years, had had symptoms for two years. On June 23, 1952, he stated that he had had pneumonia four years earlier. He had had a cough for two years. In August, 1951, after an appendicectomy he had had to return to hospital with severe upper abdominal pain. He had not much sputum. He had had no hæmoptysis but had lost some weight. The nasal sinuses appeared clear. An X-ray examination on June 12 revealed no abnormality in the chest. On June 26 a bronchographic examination revealed partial atelectasis with bronchiectasis in the left lower lobe bronchi (Figure V).

Case XXI.

A female patient, aged sixteen years, had had symptoms for eleven years. On June 5, 1952, she stated that she had had pneumonia at the age of five years and had had a cough ever since. She had a moderate amount of sputum, but had had no hæmoptysis. Bilateral antral infection was present. An X-ray examination of the chest on June 10 revealed no abnormality. A bronchographic examination on July 3 revealed bronchiectasis of the lower lobe of the left lung and the left lingular bronchus with some early sacculation; the right bronchi aroused suspicion.

Case XXII.

A female patient, aged four years, had had symptoms for four years. On August 8, 1952, the mother stated that the child was born with a cough. She had had a cough ever since. She had had a left-sided nasal discharge for years. She had undergone an operation for tonsils and adenoids twelve months earlier, and this had been followed by pneumonia. Left antral infection was present. An X-ray examination on August 11 revealed no evidence of any abnormality in the lungs or mediastinum. A bronchographic examination on November 6 revealed cylindrical dilatation of the left lower lobe bronchi. There were also some slight early changes in the right lower lobe bronchi (Figure VI).

Case XXIII.

A male patient, aged forty-two years, had had symptoms for thirty-six years. On October 21, 1952, the patient stated

that he had had a cough since he was about six years old. He coughed up more than a cupful of sputum daily. He had had no hæmoptysis, but became very breathless. He had no allergic history. The nasal sinuses appeared clear. On October 22 an X-ray examination of the lungs revealed no evidence of abnormality in the lung fields. A bronchographic examination on November 6 revealed some cylindrical dilatation of the left lower lobe bronchi. There were early similar changes in the lower lobe of the right lung.

Case XXIV.

A male patient, aged six years, had had symptoms for four years. On January 23, 1953, the mother stated that the child had had a cough since he was eighteen months old. He coughed up no sputum, but appeared to swallow some. He became breathless at times. There was a family history of allergy. The nasal sinuses appeared clear. On January 16 an X-ray examination had shown the lung fields to be within normal limits. A bronchographic examination on February 5 revealed early dilatation of nearly all the left bronchi, including those of the upper lobe. The right bronchial tree appeared normal (Figure VII).

Case XXV.

A female patient, aged eight years, had had symptoms for eight years. On April 28, 1953, the mother stated that the child had caught a severe cold at the age of two months and had had a cough ever since. At the age of eight months she had had pneumonia. She had suffered from measles at the age of two years and from whooping-cough six months later. When four and a half years old she had had pneumonitis. She produced a good deal of sputum and had had some hæmoptysis. There was a family history of allergy. A bilateral antral infection was present. An X-ray examination on April 29 showed that the pulmonary markings at the base of the right lung medially were a little more prominent than usual; but three consultants considered that the lungs were within normal limits. A bronchographic examination on April 30 revealed two or three moderate-sized cavities filled with "Lipiodol" from the right upper bronchi. There was a similar less pronounced appearance in the right mid-zone. These areas suggested a developmental defect. The left lower bronchi were broadened, but the appearance seemed more like that of an acquired bronchiectasis. The appearance of the lingula also aroused suspicion (Figure VIII).

Comment.

The usual treatment indicated has been given to these patients, such as bronchoscopic lavage, postural drainage, inhalations, breathing exercises, the administration of "Adexolin" *et cetera*, and any treatment indicated for the nasal sinuses has also been carried out. Several of these patients have undergone a lobectomy or are awaiting operation; but a number have refused surgical treatment, as they say that they now feel so much better with treatment after having waited so many years for it that they are satisfied to continue with it. The children are not quite up to lobectomy age yet, but are under consideration for later operation.

When one looks through the histories of these patients it is astounding that they could have gone for so long without a diagnosis of bronchiectasis being made. In not one of these cases had it been suggested to them that bronchiectasis was present. I wish once more to emphasize most strongly that the history of the patient must be given full consideration, no matter what the report on the plain X-ray film may be, and no matter how clear the film may appear.

After the bronchographic examination, no doubt, some slight alterations may be seen in some of the plain skiagrams in the areas affected; but the point is that these changes were not suggested without the bronchogram, and it is so easy to find them after they have been thus demonstrated. In others, I am sure, no changes will be seen even with the help of the bronchogram.

My plea is that when the history is suggestive of a bronchiectasis, though a plain X-ray examination is first necessary, no further notice should be taken of it if it fails to show any changes, but a bronchoscopic and bronchographic examination should be carried out forthwith. Only thus can we save these patients from many years of abortive treatment or even from lack of treatment and help them to recover their full, or at least much better, health.

Reference.

DAVIDSON, M. (1948), "A Practical Manual of Diseases of the Chest", Third Edition, 120.

CANCER OF THE ŒSOPHAGUS SUCCESSFULLY TREATED.

By B. S. HANSON,
Adelaide.

IT is a common medical opinion that if it is not practicable to excise a cancer of the œsophagus, all that remains to be done for the patient is a gastrostomy or insertion of a Souttar's tube. That this opinion may well be wrong is shown by the following two case reports.

Case I.

Mrs. X., aged fifty-nine years, had a barium meal X-ray examination on October 2, 1947, because she had noticed some difficulty in swallowing solid food for three months. She was able to take a full mixed diet if the food had been finely divided, but had lost her appetite to some extent. The report on the barium meal examination, performed by Dr. H. A. McCoy, was as follows:

> There is constriction of the lumen of the œsophagus, in its middle third below the level of the aortic arch. The appearances resemble an organic stricture which is likely to be associated with a carcinoma. The obstruction is not extreme and semi-solid substances passed with only a moderate delay.

On October 8, Dr. R. M. Glynn examined the œsophagus through an œsophagoscope and found a friable tumour near the middle of the œsophagus, sections of which were reported on by Dr. Eugene McLaughlin as follows:

> *Sections show a carcinoma. It is probably an* anaplastic squamous cell carcinoma.

Surgical treatment was considered and rejected by Dr. Glynn, and on October 20 the patient was referred to me for a course of radiotherapy. A mixed vitamin concentrate was ordered as a dietary supplement, and between October 21 and December 3, 1947, a dose of 5900r was given to the tumour by means of deep X rays of half-value layer 0·9 millimetre of copper, applied through six small rectangular skin fields. At the present time, six years later, she swallows without difficulty, and the site of the carcinoma can be recognized as a very slight constriction which would not appear significant without knowledge of the past history.

Case II.

Mrs. Y., aged sixty-seven years, had a barium meal X-ray examination in May, 1948, because she had noticed increasing difficulty in swallowing for four months. Her diet at that time was limited to liquids. Dr. B. C. Smeaton reported on the examination as follows:

> *There is a well-marked stricture of the œsophagus* involving the lower third. It is approximately 10 cm. in length, and allows the passage of liquids. Its appearance *suggests carcinoma.*

Dr. A. S. de B. Cocks examined the œsophagus through an œsophagoscope on May 15, and found an impassable stricture two centimetres below the bifurcation of the trachea, and examination of sections from this region showed "squamous cell carcinoma of intermediate type without cell nests" (Dr. O. Poynton). A gastrostomy was performed by Dr. I. A. Hamilton on May 21, and on June 17 the patient was referred to the Radiotherapy Department of the Royal Adelaide Hospital.

At that time she was obviously suffering from malnutrition, and it is perhaps significant that a year previously, while she was under my treatment for skin cancer, we had noted atrophic glossitis with fibrillary twitchings of the tongue and evidence of vitamin A deficiency in the skin of the hands. She had then been given dilute hydrochloric acid and vitamin supplements. A further attempt at improving the nutritional state was made, and from July 15 until August 24 a six-field course of deep X rays was given, of half-value layer 1·3 millimetres of copper, the tumour dose being 6000*r*.

Within three months there was X-ray evidence of improvement; but subsequently the lumen of the stricture became progressively smaller, and although she was maintaining her weight she was unable to swallow anything a year after her treatment. In September, 1949, Dr. Cocks again examined the stricture through an œsophagoscope, and as examination of sections from the upper and lower margins did not reveal the presence of malignant tissue, he dilated the stricture. Since then dilatation has been required every three months. After each dilatation she can swallow a mixed diet so long as it is minced, but it has been thought advisable to retain the gastrostomy for use when swallowing becomes more difficult, and dilatation is nearly due again. Now, nearly five and a half years since her treatment, she is in much better nutritional state.

Comment.

These five-year survivals after treatment for carcinoma of the œsophagus are, I believe, the first reported in Australia. Similarly successful treatments are reported from time to time in overseas clinics, and undoubtedly surgical successes will be recorded in Australia in the next few years. It is my belief that surgical excision is indicated when practicable. But of the two cases described above, in the second the condition was certainly inoperable, and in the first it would probably be so classed because of the site of the tumour. The point I wish to make is that the patient whose lesion is inoperable is not necessarily incurable even when treated with standard X-ray plants without supervoltage or rotation therapy. Of the two under discussion, the first was treated at 180 kilovolts and the second at 250 kilovolts. Moreover, a good proportion of those not cured will obtain a relatively comfortable life of one to three years, an outcome I regard as successful.

The requirements for successful treatment are a tumour which may show a little local spread but which has not developed distant metastases, a rapid improvement of nutrition by a protein-rich diet and necessary supplements, and a course of X-ray treatment in which multiple small fields are meticulously directed on to the tumour. Such patients may nearly always be improved to the extent that they can swallow a diet sufficient to maintain health, and even a poor "swallow" is better than a good gastrostomy.

Reviews.

I.

History of the Second World War: United Kingdom Medical Series. *Editor-in-Chief, Arthur S. MacNalty, K.C.B., M.D., F.R.C.P., F.R.C.S.* "Medical Research", edited by F. H. K. Green, C.B.E., M.D., F.R.C.P., and Gordon Covell, *C.I.E., M.D., D.T.M.&H., D.P.H., I.M.S.* (ret.); 1953. London: Her Majesty's Stationery Office. *10" × 6¼", pp.* 404. Price: 40s.

THIS is a notable record of the contribution by laboratory workers to the successful issue of the war. It includes practically all the officially sponsored medical research into the health problems of the fighting forces and the civil population under war conditions. Much of the work was initiated or coordinated by the Medical Research Council, with, in some instances, the collaboration of academic or commercial research laboratories; some of it, however, was promoted by the other government departments or by the fighting services themselves. A great deal of the work here described has been recorded elsewhere, frequently in other volumes of the medical history of the war, in which case the relevant notice is concise but always interesting. A list of scientific papers dealing with the work described is appended to each chapter; this includes mainly work done in the United Kingdom. The wide range of the work undertaken is truly amazing, and the successful results of research into urgent wartime medical problems are usually of much more than ephemeral value and in most cases constitute an impressive addition to the sum total of medical knowledge. The Personnel Research Committees of the various services investigated problems peculiar to their own service—for example, the Royal Navy, *inter alia*, underwater physiology and care of the shipwrecked; the army, body armour and the hazards of army fighting vehicles; the Flying Personnel Committee, the manifold problems arising from invasion of the stratosphere (in this connexion the work of Professor Frank Cotton, of the University of Sydney, is commended). The longest chapter in the book is devoted to a consideration of war wounds and injuries through the "sulphonamide era" to the extended use of penicillin—quite a masterly survey. The account in another chapter of the development of the production and clinical use of penicillin by Florey and others will be for most readers the most interesting section of the volume. In the consideration of the systemic effects of injury is included an account of the far-reaching wartime developments in investigation of the problems of blood transfusion—grouping of donors, storage of blood, natural blood substitutes, and the various complications in the recipient, the investigation of which was greatly aided by the wealth of available clinical material. The account of war diseases included a tribute to the value of work done by N. Hamilton Fairley and the Australian Army Medical Research Unit at Cairns on the prophylaxis and treatment of malaria, and to the success of Dr. R. N. McCulloch in developing the use of mite repellents in New Guinea.

The Emergency Public Health Laboratory Service proved so efficient in dealing with infectious disease and bacterial food poisoning that a similar but expanded Public Health Laboratory Service is now maintained on a permanent basis.

Many other problems of public and industrial health come under review. Wartime food rationing in its application, not only to the community at large, but also to the special needs of invalids, infants and pregnant women, was a difficult problem successfully solved by the Food Rationing Advisory Committee, which incidentally acquired a vast fund of information regarding the incidence and distribution of various diseases in Great Britain. An important result of chemical defence research was the development of BAL as an effective antidote to Lewisite gas and its subsequent successful use in various forms of metallic poisoning. Research sponsored by the Ministry of Home Security on the biological effects of explosions with special reference to air-raid casualties is reported in considerable detail, because much of it is not readily available elsewhere. An appendix to the volume contains the names of the members of the numerous wartime committees of the Medical Research Council; the wealth of talent and experience there disclosed explains the general high standard of the results achieved under the stimulus of war. The presentation is worthy of the subject, well balanced, easy to read, and interesting throughout.

II.

History of the Second World War: United Kingdom Medical Series. Editor-in-Chief, Arthur S. MacNalty, K.C.B., M.D., F.R.C.P., F.R.C.S. "The Emergency Medical Services", edited by *C. L. Dunn, C.I.E., I.M.S.* (ret.). Volume II: Scotland, Northern Ireland and the Principal Air Raids on Industrial Centres in Great Britain; 1953. London: Her Majesty's Stationery Office. 10" × 6¼", pp. 504, with 40 illustrations. Price: 50s.

THIS volume of the medical history of the war is divided into three parts. Parts I and II describe the organization of the medical service in Scotland and Northern Ireland respectively. In both instances the planning was based on the same policy and principles as in England and Wales (fully described in Volume I). There were, however, certain deviations rendered necessary by geographical considerations, distribution of population, and variation in administrative control. In both countries the planning was fully tested, as Clydeside experienced some of the most devastating raids of the war, and one raid on Belfast in particular caused very heavy loss of life. Part III, which is the largest section of the volume, describes raids on London and on various industrial cities and seaports, as well as the so-called "Baedeker" reprisal raids on cathedral cities. This is absorbingly interesting. To make the problem of civil defence quite clear, extracts are quoted from the reports

and comments of medical officers of health and group sector officers which give a very vivid picture of the difficulties and dangers of rescue work. This, for example, from Liverpool:

> All the work of collecting the casualties at incidents, rendering essential first aid, transferring them to first-aid posts and hospitals, and from first-aid posts to hospitals, had to be carried on in the dark, hour after hour. The first-aid services had to find their way to incidents mostly by circuitous routes, because roads were blocked by craters and debris, and they had to collect casualties, mostly from ruined buildings, and buildings made difficult of access by direct hits and blast. Tunnels through unstable materials had to be dug in order to release trapped casualties. Ambulances had to find their way through streets partly or wholly blocked; the personnel of first-aid posts had to deal with hundreds of casualties often in partly wrecked buildings with blackout rendered ineffective, by the light of torches and lanterns and to make quick decisions as to priority of treatment, or dispatch to hospital of those who required more expert attention.

In the hospitals the staff worked at high pressure throughout the night, frequently under makeshift conditions, and with daylight came the necessity to evacuate patients to make room for possible casualties of the next night. Many of the hospitals were damaged by bomb and blast, frequently with severe loss of life.

Frequent interruption of water supply with occasional contamination by sewage caused fears of an epidemic of typhoid fever; but rapid repair of the damage and immediate institution of an alternative supply, usually with chlorination, averted the danger. Infectious disease did not in fact at any time present unusual problems. The splendid reports quoted are quite matter of fact and without any deliberate emotional appeal, but it is impossible to read them without being stirred by the dauntless courage under most trying conditions of all those in all ranks of life who were engaged in civil defence; by the patience and fortitude of the casualties themselves; and by the high morale of the general population and their grim determination to see the business through to a victorious end. Many deeds of high valour are recorded, but many more must necessarily have escaped notice. It is difficult for the ordinary citizen to discover what is happening in Australia about civil defence; but if the Government is really taking up the matter seriously, here, in this account of civil defence in action, will be found an answer to most of their problems. However, the appeal of this volume is to a much wider circle as a stimulating and inspiring account of Britain's survival against overwhelming odds.

Modern Trends in Diagnostic Radiology (Second Series). Edited by J. W. McLaren, M.A., M.R.C.P., F.F.R., D.M.R.E.; 1953. London: Butterworth and Company (Publishers), Limited. Sydney: Butterworth and Company (Australia), Limited. 10" x 7", pp. 424, with 359 illustrations. Price: £4 15s. 6d.

THIS book contains contributions from various well-known radiologists in Great Britain and the Continent. The design of apparatus is discussed and one is rather staggered by the elaborate type of apparatus and its great cost. The cost of this specialized gear places it beyond the reach of private practitioners and allows of its installation only by large institutions.

The camera type of apparatus for mass radiography is being replaced by the Schmidt mirror units, which are quite efficient and less costly.

It is rather surprising to see that tomographic (body section) radiography is increasing in popularity in Europe. Image intensification work is generally becoming more popular, but its cost is still prohibitive. The use of multiple exposure in cardioangiography, angiography and cineradiography has increased greatly the risk of damage to patient and operator and caution is urged in this type of work.

An interesting chapter on fungous disease of the lungs with good illustrations of such lesions as actinomycosis, aspergillosis, torulosis *et cetera* is of great value. Histoplasmosis, uncommon in Australia, is a major cause of the scattered calcified areas so often seen in films. It is confined mainly to the American continent and parts of Asia. Angiocardiography is covered in a clear fashion and is well illustrated. The small intestine is receiving greater attention, and interesting work is discussed on the rate of bowel movement and on the changes in the mucosal pattern. The presence of mucus often produces flocculation of the barium mixture and causes errors in interpretation. Pancreatitis is being more frequently recognized and some good illustrations are shown of this condition and of malignant changes in the pancreas.

Arthrography, especially of the knee and shoulder, has shown useful advance. Disk protrusion receives attention and it is pointed out that the spinal cord fills the canal in the cervical region more completely than in the other spinal regions and consequently symptoms are more likely to occur from slight disk abnormalities in this region. The section on cerebral angiography is quite up to date and again the reader is struck by the necessity of specialized skill in the radiologist. The illustrations of renal angiocardiography are excellent. The book ends with an informative chapter on the legal aspects of radiological work.

In all, this is an excellent work and worthy of detailed study.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Water, Electrolyte and Acid-Base Balance; Normal and Pathologic Physiology as a Basis for Therapy", by Harry F. Weisberg, M.D.; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9¼" x 6", pp. 262, with nine illustrations. Price: £2 13s. 9d. from The Williams and Wilkins Company.

The basis of this book was a seminar in general medicine conducted at the Mount Sinai Hospital of Chicago.

"Aids to Diagnosis and Treatment of Diseases of Children", by F. M. B. Allen, M.D., F.R.C.P. (Lond.), and O. D. Fisher, M.D., M.R.C.P. (Lond.), D.C.H.; Ninth Edition; 1953. London: Baillière, Tindall and Cox. 6¾" x 4", pp. 308. Price: 8s. 6d.

One of the well-known Students' Aids Series.

"Disorders of the Blood: Diagnosis, Pathology, Treatment, Technique", by Sir Lionel E. H. Whitby, C.V.O., M.C., M.A., M.D. (Cantab.), F.R.C.P. (Lond.), D.P.H., and C. J. C. Britton, M.D. (New Zealand), D.P.H.; Seventh Edition; 1953. London: J. and A. Churchill, Limited. 9¾" x 6¼", pp. 870, with many illustrations, some in colour. Price: 63s.

The first edition was published in 1935 and the sixth in 1950.

"Anoxia of the New-Born Infant", a symposium organized by the Council for International Organizations of Medical Sciences established under the joint auspices of U.N.E.S.C.O. and W.H.O.; Consulting Editors, Dr. Kenneth W. Cross (U.K.), Dr. Marcel Lelong (France) and Dr. Clement A. Smith (U.S.A.); Editors for the Council, J. F. Delafresnaye (France) and T. E. Oppé (England). Oxford: Blackwell Scientific Publications. 8¾" x 5¾", pp. 248, with many illustrations. Price: 27s. 6d.

The symposium was held early in October, 1951; the first printing took place in September, 1953.

"Peptic Ulcer", by C. F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S.E., F.R.F.P.S. (Glas.); 1953. Edinburgh and London: E. and S. Livingstone, Limited. 9¾" x 7", pp. 296, with many illustrations, some in colour. Price: 42s., postage abroad 1s. 3d.

The author has brought together the more important elements of present-day knowledge on peptic ulcer and presented them in the form of a monograph.

"The Hallucinogenic Drugs (The Insanity-Producing Drugs: Indian Hemp and Datura): A Neglected Aspect of Forensic Medicine (A Loophole in the Law)", by Donald McI. Johnson, M.A., M.B., B.Ch., Barrister-at-Law; 1953. London: Christopher Johnson. 8½" x 5½", pp. 46. Price: 5s.

The author describes an experience of his own and draws important conclusions from it.

"New Light on Dreams: A New Approach to the Dream Problem", by Max Serog, M.D.; 1953. Boston, U.S.A.: The House of Edinboro, Publishers. 8" x 5¼", pp. 160. Price: $3.00.

The author attempts "to make understandable dream character and dream phenomenology and to illuminate the various aspects of the dream".

The Medical Journal of Australia

SATURDAY, MARCH 13, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE TENTH RHEUMATISM REVIEW.

THE appearance of the Tenth Rheumatism Review by the Editorial Committee of the American Rheumatism Association should not pass unnoticed in this journal. The First Rheumatism Review was published in *Annals of Internal Medicine* in the year 1935. The initiation of the rheumatism reviews and their continued appearance since 1935 have been made possible chiefly because of the energy and devotion of Dr. Philip S. Hench. The Ninth Review was published in 1948. All the reviews have appeared in *Annals of Internal Medicine*. The Ninth Review was concerned with the American and English literature of 1941 "through 1945", and was published in February, 1948. The Tenth Review is concerned with the literature from 1946 "through 1950"; it is appropriately dedicated to Dr. Philip S. Hench. The Tenth Review appears in the issues of September and October, 1953, of *Annals of Internal Medicine*. It covers some 250 pages of the journal, and concludes with 2256 references (22 are from THE MEDICAL JOURNAL OF AUSTRALIA). These references are in alphabetical order and are complete in every respect with the full title of the several articles. It will be clear at once that it will be impossible in the space available to attempt an account of the contents of the review in any detail. The review itself is a summary of work that has been done in many places, and here and there throughout are editorial comments, for which the editorial committee alone is responsible—they do not represent the views of the American Rheumatism Association. The chairman of the editorial committee is William D. Robinson, of Ann Arbor, Michigan. He has on his committee some eleven members, whom it is not necessary to name. Our chief object in drawing attention to this review is to let readers know that it has appeared, and to give them some idea of the enormous field that is covered by it.

In the opening paragraph the definition of rheumatism and rheumatic diseases is discussed. It is pointed out that the terms rheumatism and rheumatic diseases are now properly used to refer to a large group of diseases of the muscular and skeletal system characterized by pain and stiffness of joints, muscles and related structures. These disorders affect the various articular structures (cartilage, joint capsule and bone), the connective tissue around them and in some instances the fibrous tissue in a wide variety of organs. Rheumatism has been used as a collective term for illnesses in which pain in or about the joints has been a prominent symptom. The enormous ground covered will best be indicated by enumeration of the main headings of the two parts into which the review is divided. In Part I we have the general incidence of rheumatic diseases, classification of diseases of joints and related structures, diseases related primarily to trauma, arthritis due to specific agents, the adrenal cortex in rheumatic diseases, rheumatic fever, rheumatoid arthritis, rheumatoid (ankylosing) spondylitis, osteoarthritis, backache and sciatica, gout and gouty arthritis, and the uric acid problem. Under arthritis due to specific agents the subjects dealt with include gonorrhœal arthritis, tuberculous arthritis, syphilitic arthritis and synovitis, yaws, meningococcic arthritis, suppurative arthritis, typhoid arthritis, brucellosis, rat-bite fever, the rarer forms of specific infectious arthritis, and mycotic diseases of joints. In Part II we have "collagen diseases", psoriatic arthritis, Reiter's syndrome, neuropathic arthropathies, epidemic tropical acute polyarthritis, hæmophilic arthritis, rheumatic purpura, palindromic rheumatism, allergic arthritis, pharmaceutic arthritis, alkaptonuria and ochronosis, the painful shoulder, reflex sympathetic dystrophy, brachalgia, monarticular rheumatism, tumours of synovia, arthritis related to primary bone disease, congenital defects, structure and function of articular tissues, experimental arthritis and the campaign against rheumatism. It is not surprising to read under the heading of general incidence of rheumatic diseases that among the chronic diseases rheumatism had ranked first in incidence and second only to nervous and mental disorders as causes of disability. It was estimated (we must remember that this refers to the period 1946 to 1950 inclusive) that in the United States more than 300,000 persons were made unemployable by rheumatic diseases every year, and that there were more than 147,000 rheumatic invalids; also, the diseases accounted for more than 92,000,000 days lost from work each year, representing an economic loss of half a billion dollars a year. It is interesting that in Scotland during the year 1937-1938 there were over 45,000 new cases of incapacitation from rheumatism in the insured population of 1,837,000. In two localities in Scotland 7% and 9·4% of all visits for medical care were because of rheumatism. In Great Britain 7% of the population were incapacitated by rheumatism for at least one day each year, and half of the population suffered from rheumatism which was not disabling. We know of no figures for the incidence of rheumatism in Australia. Turning to rheumatic fever, we read that approximately 2% of school children in the United States have rheumatic heart disease, that 90% of all heart disease occurring in persons below the age of twenty is due to rheumatic fever, and that rheumatic fever annually causes more than ten times as many deaths

as poliomyelitis. Would that there was as much concern about the prevention of rheumatic fever or about research into it as there is in regard to poliomyelitis. Perhaps we have grown used to rheumatic fever and look on it as part of our normal heritage. This is surely a wrong attitude to adopt. The editor of this review adds at this point that most observers are agreed that the incidence and actual severity of rheumatic fever are decreasing, but that no accurate figures are available because of inadequate reporting or registration systems in most parts of the world. If this decreased incidence is a fact, it would be interesting to know how much of it is due to the widespread use of sulphonamide drugs for all kinds of complaints. As a matter of fact, there is a great deal about rheumatic fever in this report which might well be made the subject of a separate discussion.

Since Hench has been so prominently associated with the discovery and use of cortisone and ACTH, and since this report is dedicated to him, it will be suitable to refer to the section in the report dealing with the adrenal cortex and rheumatic disease. The mode of action of cortisone and ACTH in rheumatic diseases is discussed, and it is stated that the search for the mechanism by which they produce their dramatic suppression of rheumatic diseases has taken many directions. Their use in rheumatoid arthritis was originally suggested by the observed potentiality and natural remissions in the disease, particularly the association with pregnancy and jaundice. We read that subsequent investigations have neither established nor disproved that adrenal cortical hormones are the common denominator in spontaneous remissions. While there is some evidence of increased adreno-cortical activity during the last three months of pregnancy, no claim has been made that this explains remissions occurring during pregnancy; there is even less factual information about the hormonal status during jaundice. Sayers declared that the adrenal physiologist was at a loss to give a rational explanation of these empirical discoveries. The inhibition of hyaluronidase action is discussed. In this discussion we are told that the permeability of membranes and of synovial membrane appears to be increased by DCA (11-desoxycorticosterone) and hyaluronidase; the effect of hyaluronidase appears to be antagonized by cortisone and several other steroids. The editor adds that several studies, including some reported since the period covered by the review, have shown unequivocally that adrenal cortical extract as well as ACTH and cortisone does not alter the permeability of the normal joint membrane or any other tissue that has been tested. However, when the permeability has been increased by hyaluronidase administration or by inflammation, then the 11-oxysteroids and ACTH tend to reduce the permeability towards normal. He states that the possibility that a reduction in permeability is an important common denominator in the anti-inflammatory action exercised by cortisone and ACTH has been emphasized recently by several writers. The important point which arises from the discussion of this aspect of the review from the practical point of view is that nearly all the untoward effects encountered in the use of the agents in question for a few weeks or even a few months can be handled by regulation of dosage and by simple adjuvant measures dependent on careful clinical observation. The editor adds that with attempts to use these hormones to control chronic diseases over a period of years the possible production of osteoporosis, development of peptic ulcer, fear of masking intercurrent infection or disease, and the attainment of a balance between undesirable effects and reasonable control of disease present problems which have not yet been solved.

In the list of the sections discussed in the review there appears the expression "pharmaceutic arthritis". Readers will naturally wonder what this can mean. Here the committee refers to joint manifestations related to the use of drugs or other therapeutic agents which give rise to sensitization. Some of them, we are told, furnish the most convincing examples recorded of allergic arthritis.

In the reports of societies and congresses held in different parts of the world the expression is sometimes seen that an article by an author is a report "by title only". We fear that this reference to the Tenth Rheumatism Review has been almost by title only, but if the interest of readers has been aroused even in its existence our purpose will have been achieved.

Current Comment.

A DOUBLE CENTENARY.

MARCH 14 and 15, 1954, will be the hundredth anniversaries of the birthdays of Paul Ehrlich and Emil von Behring. These anniversaries will be celebrated from March 13 to 16 at Frankfurt-am-Main and at Marburg-am-Lahn.

As pointed out in the obituary notice of Ehrlich, published in this journal in September, 1915, Ehrlich's name is always associated in the public mind with the discovery of salvarsan or "606". This was, of course, an important discovery, but it was not sufficient to justify the enshrinement of Ehrlich's name as one of the greatest scientists of his day. Sir Henry Dale, in his introduction to Martha Marquardt's biography of Paul Ehrlich, writes that the public have come to regard Ehrlich as the man who discovered salvarsan, and he adds that not so many are aware that nearly twenty years earlier the possibility of making practical use as remedies of the substances, antitoxins *et cetera* formed by the body's natural reactions of immunity was made effective by Ehrlich's masterly researches. He goes on to state that the major credit for that achievement, as not seldom happens, had been given to others, such as von Behring, who had made the primary and relatively easy observation that such natural antidotes are formed. It was Ehrlich, he declares, who showed how their production could be so stimulated that they could be obtained in sufficient strength for practical use in treatment; and it was he who devised accurate methods for measuring their purity and potency in terms of permanent standards, and defined units of activity in which the values could be expressed. Von Behring's name is best known by his work on diphtheria. It was he who first produced antidiphtheritic serum, and began to produce it upon a grand scale—this was in 1894. We all know that this type of serum is the specific treatment for diphtheria. It is interesting to observe that both Ehrlich and von Behring received a Nobel prize for physiology and medicine. Von Behring received the prize in 1901, and Ehrlich received it in 1908 jointly with Metchnikoff, of the Pasteur Institute of Paris. As occasionally happens between great men who work together, relations between Ehrlich and von Behring at one stage became very strained. It was the same, we remember, with W. S. Gilbert and Arthur Sullivan. Martha Marquardt writes about the subject at considerable length, and states that von Behring exploited Ehrlich. Though official relations continued between them, friendship

apparently had ceased. Marquardt records that on August 20, 1915, when Ehrlich died, von Behring came to Frankfurt to take part in the funeral ceremony. He followed the funeral procession with some difficulty, as he was an aged, sick man. He had written a touching obituary notice which he published later on, but in the face of death at the open tomb he stammered the following words (they are translated from the German): "Now you are at rest, my dear friend, you always had a sensitive soul, and if we have hurt you, forgive us."

THE DERMAL MIRROR.

THE reflection of emotions in the skin is a commonplace phenomenon. The blush of shame, the pallor and sweating of fear, the bristling of alarm (more dramatically evident of such animals as the cat), these are as eloquent of the individual's feelings as facial expression and much more reliable because of their involuntary character. It is natural enough that current interest in the psychosomatic concept of disease should have directed attention to the emotional factor in skin diseases and their possible psychogenic origin. Many papers have been published on the subject, some of them highly speculative, and opinions on the subject are by no means in agreement. This is explained, in part at least, by the fact that the subject concerns members of two highly developed specialties, and trained psychiatrists are seldom skilled dermatologists or skilled dermatologists trained psychiatrists.

A reasonable account of one view was given by G. M. Frumess[1] at a meeting of the American Medical Association in December, 1952. In this, he examines the emotions responsible for changes in the skin, describes the physical agencies through which they mediate these effects and discusses diseases of the skin that appear to have important emotional components. In a consideration of the emotional components of psychosomatic dermatosis, Frumess refers to the unsatisfied hunger for love, to anxiety, to hostility and to the sense of guilt. He points out that the need for love is one of humanity's greatest hungers. If this hunger is unsatisfied, the resulting frustration may lead to other unpleasant emotions. The person who believes himself unloved may resort to symbolic or perverted ways of obtaining affection. If these substitutes overflow into the autonomic nervous system, itching and scratching follow. Thus, certain skin diseases, especially localized neurodermatitis and ano-genital pruritus, may be "unconscious erotic substitutes for a lack of love". He quotes the statement of Yaskin that anxiety is the central symptom of nearly all psychoneuroses. If the causes of anxiety are below the level of consciousness, an overflow into autonomic pathways commonly occurs; and bodily dysfunctions, including skin disorders, may result. Turning to the physiological factors in emotions and dermatoses, Frumess points out that the emotions may cause changes in the skin by releasing certain substances at the autonomic nerve endings. These substances in turn may act synergistically with other biological factors to cause alterations in the skin. Both the sympathetic and the parasympathetic divisions of the autonomic nervous system have physiological connexions with the higher brain centres and may, therefore, be stimulated or inhibited by psychic changes. Parasympathetic fibres in general and sympathetic fibres to sweat glands liberate at their terminals acetylcholine, which causes vasodilatation and secretory activity of the oil glands and sweat glands. Excitement, hostility and embarrassment may stimulate these fibres with subsequent flushing of the skin, oil secretion and sensations of itching. Release of more acetylcholine causes further permeability of the blood vessels, whereupon wealing or hives may occur; still further permeability results in the escape of formed elements of the blood with consequent purpura and even frank bleeding. Sympathetic fibres, stimulation of which is seen in reactions to fear and anxiety, liberate an adrenaline-like substance called sympathin, with production of pallor, coldness, sweating, *cutis anserina* (goose-flesh) and crawling sensations. Some emotions produce a mixture of sympathetic and parasympathetic effects on the skin. Frumess suggests that there is a special relationship between allergy and the nervous system which is explained biologically in the synergism of acetylcholine and histamine. By this he explains why many people experience allergic reactions only when they are emotionally disturbed. Discussing the role of emotions in specific diseases, Frumess states that emotional factors affect two skin diseases of proved organic ætiology—namely, warts and "fever blisters". It is well known that warts can be cured by suggestion. Recurrent *herpes simplex*, as manifested by "fever blisters" or "cold sores", has a proved virus ætiology. The latent virus carried by most adults is usually activated by upper respiratory tract infections, fever and other identifiable factors, including emotional disturbances.

Summing up, Frumess states that commonly encountered emotions, such as insecurity, anxiety, hostility and guilt, may cause stimulation of the autonomic nervous system, leading to flushing, sweating, itching and excoriating. Physical agents through which emotions mediate these effects are acetylcholine released at parasympathetic nerve fibre endings, sympathin produced by sympathetic stimulation, synergism between acetylcholine and histamine, and the mechanical and allergic effects of sweat retained under pressure. Several skin disorders believed to be of emotional origin are related to fairly characteristic personality patterns. Frumess seeks to relate the clinical entity to the emotional pattern in cases of *herpes simplex*, rosacea, urticaria, childhood eczema, adult neurodermatitis and localized neurodermatitis. He considers that in most such cases it is desirable that one medical adviser should supervise both the somatic and psychic aspects of therapy. With functional dermatoses resulting from deep-seated neuroses or psychoses, the patient should be referred to the psychiatrist for treatment.

It is as well to point out that many experienced dermatologists, while recognizing that emotional responses have profound effects upon the skin, view some of the ideas of the psychosomatic school with caution, if not with frank scepticism. An informed exponent of this attitude is Marion B. Sulzberger, whose careful discussion of the question in "The 1951 Year Book of Dermatology and Syphilology" provides a healthy corrective to the more extreme enthusiasm for the psychosomatic fashion. He suggests that it is not astonishing that dermatology should offer "a fallow field" for psychosomatic speculations, for there are many skin diseases of unknown cause, and it appears to be particularly distressing and abhorrent to both patients and physicians to confess to ignorance in regard to a disease which is fully exposed to the light of day. It is an easy way out to ascribe almost any skin diseases of unknown ætiology and erratic course to the emotional fluctuations and changing tensions which are the essence of all life. It saves the confession of ignorance in relation both to diagnosis and to treatment, and it saves the considerable amount of time and money which may have to be expended on arduous, lengthy and costly tests, which, though indicated, may not produce positive information of value. However, Sulzberger states that to conclude without strict proof that any skin disease or cutaneous symptom is "psychogenic" or is due to tensions, fatigue, ambitions, cravings and disappointments will not be likely to cure the sufferer and will inevitably retard the discovery of the true causes of obscure dermatoses and the advancement of medical knowledge. Much thinking on the subject is loose and characterized by failure to eliminate *post hoc, propter hoc* reasoning and to "pay due respect to the trap of the 'cart-horse' fallacy". Sulzberger emphasizes the error of thinking of any somatic disease or reaction as purely and exclusively somatic, or of any psychic disease as purely and exclusively psychic. Psyche and soma, he states, together form a single unit; they are so integrated, so inextricably dovetailed and correlated that it is only in combination that they form the functioning organism. No psychic change can possibly fail to exert some effect on the body and the bodily functions, and

[1] *J.A.M.A.*, August 8, 1953.

no bodily change can take place without some effects on the mind and emotions. The careful, critical and intelligent study of the skin and its responses has always afforded one of the best opportunities for investigating just such delicate relationships and intricate reciprocal effects between different organs and systems. Sulzberger emphasizes the importance of dermatologists (and all others with special knowledge concerning the skin organ) studying the possible reciprocal effects between the psyche and that organ, and offers two main reasons: firstly, properly evaluated expert studies of the skin offer perhaps the finest opportunities for testing, proving or disproving many of the prevalent ideas concerning psychic effects on somatic disease; secondly, whenever the pressures and temptations to accept a particular theory are strongest, the scientist (here the trained dermatologist) will redouble his efforts to study critically, to experiment carefully and to weigh objectively. He puts forward a list based entirely on his own experience of skin diseases and skin reactions which have been observed to respond promptly and unequivocally to the influences of the psyche and the emotions, as well as some of those common skin diseases and reactions in which so far, despite deliberate and repeated efforts, indubitable direct effects of psychic and emotional influences have not been observed. He points out that these observations fall short of those of many of his colleagues in the number of skin phenomena and diseases observed to be clearly and directly influenced by the psyche and the emotions; but although he lays no claim to infallibility, he expresses his conviction that at present many hasty and inaccurate conclusions are being drawn in this regard. He points out the danger of using misleading terminology, such as neurodermatitis, which cannot be justified on the basis of observed facts, and the temptation of the dermatologist to take the line of least resistance. He expresses this last-mentioned situation graphically:

> At the end of a long, tiresome day, a day filled with the inevitable succession of admissions of ignorance, and the frustration of repeated failures to relieve or to cure, have not all of us—dermatologists and non-dermatologists alike—sometimes been sorely tempted to answer the insistent questions of a poor sufferer who is desperately grasping for some straw of certitude and solace, by replying: "Well, perhaps your nerves are giving you this skin trouble. Take a rest; go away for a rest—go away and let both of us have a rest!"

In this insidious fashion, he comments, begins the physician's descent on the easy path of glib phrases and away from the hard roads of detailed examination, sharp thinking and kind but accurate answers. The sad truth is that medicine does not yet know the causes of most diseases, including many skin conditions. Although emotional and psychic factors can play some part and must be weighed in conjunctfon with all other influences in almost any skin disease, they are, in Sulzberger's experience, no more likely to be of primary importance than friction, humidity, heat, cold, secondary pyogenic or mycotic infections, or the multitudinous other adjuvant factors which are contributory to the fluctuations of so many dermatoses. *Mutatis mutandis* this statement could be applied in many other fields of medicine and should be so applied, not to eliminate the psychosomatic concept from medical thinking, but rather to allow it to find its true and valuable place.

STERILIZATION PROCEDURES IN HOSPITALS.

By direction of the Honourable the Minister for Health and Home Affairs of Queensland, a "Handbook of Sterilization Procedures" has been produced.[1] This book is the work of an advisory committee, of which Dr. D. W. Johnson, the Deputy Director-General of Health and Medical Services, was chairman. The Director-General of Health and Medical Services (Dr. A. Fryberg) was a member of the committee, and the other members included Dr. A. E. Lee, Dr. R. A. O'Brien, Dr. A. D. D. Pye, Dr. E. A. Shaw and Dr. J. I. Tonge. The book is intended primarily for use in Queensland public hospitals, but it is drawn up on such practical lines that it should be useful in many other places in Australia.

Attention was first directed to the method of sterilization used in Queensland hospitals, public and private, by the occurrence of several cases of neonatal and post-operative infections caused by spore-forming organisms. Sterilization techniques were found to vary from hospital to hospital. In a few instances the procedures used were of undoubted efficiency. Some hospitals used efficient methods for some procedures, only to fall down on others, while other hospitals used sterilization techniques which were "uniformly unsafe". Very often the sterilization procedures were carried out by persons who did not understand the principles of the procedures. The extreme difficulty of killing spore-forming bacteria was not understood, and the temperature and exposure necessary to produce sterility in various items commonly used in hospitals were not appreciated. Some hospitals did not possess autoclaves; others had autoclaves which were unsafe; others again had efficient autoclaves which were used in an inefficient way. In general undue reliance was found to be placed on sterilization by chemicals, in spite of the fact that chemical disinfectants rarely produce sterility. In the foreword the sage remark is made that the rarity of infection with pathogenic spore-forming organisms is due not so much to the efficiency of sterilization techniques generally adopted as to the comparative rarity of their natural occurrence in articles brought into contact with human tissues. "Heavy expenditure has been incurred in replacing or repairing inefficient hospital autoclaves in Queensland, but the price of safety cannot be assessed in money."

Successive chapters deal with the history of heat sterilization, a review of methods of sterilization, the testing of autoclaves, sterilization by moist heat (autoclave), sterilization and care of surgical instruments, sterilization and care of syringes and needles, sterilization of miscellaneous items, chemical disinfection, and hygiene in the operating theatre. The last chapter is a summary of sterilization methods.

It is clear that the issuing of a book on sterilization methods for use in Queensland hospitals will not be sufficient to secure the use of the methods. In the long run the members of the medical profession who attend hospitals, particularly those who undertake surgical operations, are responsible for the methods of sterilization in use. This is a matter on which all practitioners of medicine who have to rely on hospital sterilization should refresh their memories. It is to be hoped that the Queensland Department will meet with success from this publication, and that it will be used in other parts of the Commonwealth.

[1] "Handbook of Sterilization Procedures: Recommended Techniques for Use in Queensland Public Hospitals", by members of the Advisory Committee on Sterilization Procedures: 1953. Brisbane: Issued by direction of The Honourable the Minister for Health and Home Affairs. 8½" × 5½", pp. 116, with 30 illustrations. Price: 7s. 6d.

IPRONIAZID IN THE TREATMENT OF BONE AND JOINT TUBERCULOSIS.

IPRONIAZID (1-isonicotinyl-2-isopropylhydrazine) is an isopropyl derivative of isonicotinic acid hydrazine, more usually described these days as isoniazid. It has been in use for some three years in America, first on an experimental basis and then on a restricted sale basis, and a preliminary assessment of its value is now due, as various reports that have appeared indicate that it displays notable activity in the treatment of patients suffering from tuberculosis. In an editorial note in "The Year Book of Medicine (1953-1954 Year Books Series)" Carl Muschenheim states that the dramatic symptomatic improvement observed in early clinical studies of the hydrazine derivatives of isonicotinic acid are to be seen more frequently with iproniazid than with isoniazid. However, he continues, iproniazid is many times more toxic

than isoniazid; and in evaluations of the therapeutic effectiveness of the two drugs there have been few indications that iproniazid is superior with respect to such objective criteria as rapidity and extent of radiological improvement or elimination of bacillary output in the sputum. Muschenheim's view is that whatever therapeutic advantage there may be in favour of iproniazid is outweighed by its greater toxicity. On the other hand, D. M. Bosworth, H. A. Wright, J. W. Fielding and H. J. Wilson,[1] who have had extensive experience of iproniazid during the period of its availability, believe it to be for the present the drug of choice in the treatment of tuberculous bone and joint lesions and are not deterred by its disadvantages, although they state frankly that patients under medication exhibit multiple evidences of toxic effects. Their experience indicates that all these are mild or reversible and appear unimportant except, possibly, psychoses; and psychoses are rare, can probably be avoided by dosage reduction and also seem to be reversible. Their statement is that iproniazid, like any chemical if active in the treatment of a disease, can and frequently does cause toxic symptoms, some of which may be severe. Complete knowledge about benefits arising from its use as well as toxic effects, both immediate and delayed, is not yet available. Much more time must elapse before there is certainty about effective dosage and a means of avoiding toxicity. Isoniazid, as contrasted with iproniazid, has been reported as showing a lesser number of toxic symptoms and a greater range of safe dosage. The feeling (that is their word) of Bosworth and his colleagues, based on seven cases in which isoniazid was used, is that this may be true in relation to minor toxic symptoms, but not to major ones (liver damage and psychosis). They feel certain that isoniazid has a much less rapid and specific effect where bone and joint lesions are concerned than has iproniazid. They report their experience with iproniazid in the treatment of 66 patients suffering from 98 tuberculous lesions of bones and joints. Of the 98 lesions, 45 were healed. In 37 improvement was noted, and in 16 there was no improvement. Five patients died while under treatment with iproniazid, but in none of these was there evidence of iproniazid toxicity. Analysis of data about the 66 patients as distinct from the 98 lesions showed that 53 had had some amount of streptomycin before iproniazid therapy was begun. In all cases, not only streptomycin therapy but all other medication was stopped at the time of beginning the iproniazid therapy. Twenty-three of the 66 patients underwent operative procedures; no complications were encountered, and wound healing was unusually good. The position at the time when the paper was presented was that, of the 66 patients, 29 had undergone healing, and medication had been discontinued. Five were healed, but were still receiving medication, 20 were "improved" and were still receiving medication, and three were improved but had been taken off medication. Five had died (these have already been mentioned) and one would probably die. Two had had less than a week of treatment. One patient had remained entirely without improvement. Assessment of radiological changes indicated that generalized osseous decalcification after several weeks of medication still seemed to occur. Bone proliferation or hastening of surgical ankylosis did not seem to occur. Actually, there was no noteworthy trend in relation either to increasing or to decreasing osteogenesis.

The numerous toxic effects are examined in detail in this paper. Bosworth and his colleagues state that the only such effect in which evidence of real danger appeared was the occasional development of a psychosis. All other effects were temporary and usually not severe; they included dizziness, tremor, Babinski response, hypertonic reflexes, clonus, euphoria, drowsiness, headache, tinnitus, coma, mouth dryness, diaphoresis, visual accommodation weakness, hesitancy, obstipation, impotence, flushing, muscle twitching and paræsthesia. Six patients developed definite psychosis; in two cases it was mild, in one it was moderate and in three it was severe. The average time of onset of psychosis was after one hundred and forty-two days of medication with a range of forty-five to three hundred days. The average duration of the acute psychotic state was thirteen days with a range of from one to thirty days. In one case iproniazid therapy had suddenly been withdrawn, and the psychosis developed three days afterwards. At the time when the paper was published all six patients had completely recovered. Although they are not daunted by the rather formidable list of complications, Bosworth and his colleagues emphasize the necessity for patients to be treated in hospital for two to four weeks during the establishment of medication, even when admission to hospital is not required on other grounds, and they also point out the need to combine iproniazid therapy with good medical care, dietary care and surgery when indicated. They make the statement, which they have never before felt that they could make, that it may be that by the use of this drug in children a tuberculous weight-bearing joint may be spared arthrodesis. They point out that in cases of tuberculosis with severe or multiple involvement, a percentage of mortality and failure must be expected; and while they readily agree that iproniazid is not a "miracle drug", they state that it is well worth continued use and investigation. Further planned study of the comparative action of iproniazid and isoniazid carried on without bias is essential. Discarding their impressions of the past, Bosworth and his colleagues plan in the immediate future to treat a contrasting series of ten patients with isoniazid, expecting that with the experience of iproniazid already gained they will be able to obtain a much fairer evaluation of the comparative action of isoniazid. Reports of their further work will be awaited with interest. At least until these and other reports appear, it would not be wise to bring iproniazid into general use.

[1] *J. Bone & Joint Surg.*, July, 1953.

THE PREVENTION OF AIRSICKNESS.

THE use of drugs in the prevention of airsickness and seasickness is the subject of perennial comment, but the importance of the problem justifies reference to recent tests carried out at the USAF School of Aviation Medicine, Randolph Field, Texas, by H. I Chinn, L. J. Milch and A. J. Dugi.[1] The drugs tested were scopolamine, the antihistamine promethazine hydrochloride ("Phenergan") and a long-acting antihistamine prepared in Belgium named "Postafene". In the test 20 young airmen were exposed simultaneously to an hour's flight of standardized turbulence. Each received a capsule twenty-four hours before flight and a second, of identical appearance, one hour before flight. Those receiving "Postafene" had the drug first and a placebo later; for the "Phenergan" and scopolamine tests the order was reversed. The placebo group received placebos on both occasions. The effect on the subjects was observed from objective and subjective reports. Each of the drugs afforded significant protection when compared with the placebos. No difference could be detected between "Postafene" given twenty-four hours in advance and "Phenergan" given one hour before the take-off. Scopolamine, however, afforded better protection than either of these drugs. Side effects were slight, the most significant being an increased incidence of dry mouth after the taking of scopolamine. Summed up, the most striking findings in this study were the excellent protection offered by the long-established drug scopolamine and the long duration of action of the antihistamine "Postafene". Chinn, Milch and Dugi state that from the low incidence of side effects, it would appear that a single dose of one milligramme of scopolamine is not excessive. However, they point out that when scopolamine must be given three or four times daily for several days, visual and nervous disturbances may sharply limit its usefulness. This limitation is likely to be less significant in relation to airsickness than to seasickness, for which scopolamine appears otherwise to be very useful.

[1] *J. Appl. Physiol.*, October, 1953.

Abstracts from Medical Literature.

PÆDIATRICS.

Changing Patterns of Respiration in Newborn Infants.

H. C. MILLER AND F. C. BEHRLE (*Pediatrics*, August, 1953) studied the respiratory activity of quiet and sleeping newborn infants by pneumographic tracings and by clinical observation. Four successive stages of respiration were noted. The first lasted only a few minutes after birth. The second lasted from a few hours to a day or more. In this stage, respiratory activity was characterized by a wide range of rates, regular rhythm and "synchronous" respiratory pattern. In this pattern, chest and abdomen contracted evenly and together. The third stage lasted several days or weeks. During this period variable rhythms and patterns of respiration were observed. Two common patterns were described as "simple retraction" and "see-saw". In the simple "retraction pattern", the lower part of the chest wall was drawn in as the abdomen and upper part of the chest expanded. In "see-saw" breathing, the whole chest wall moved in and down as the abdomen expanded. The fourth stage was characterized by a return to more stable rhythms, and only "synchronous" and "simple retraction" patterns of breathing occurred. The absence of retraction in stage two was associated with an excellent prognosis. The appearance of any form of retraction, except the "see-saw" pattern, in the first twenty-four hours, presaged serious difficulties for the infant.

Meconium Peritonitis.

WILLIAM L. BENDEL, JUNIOR, AND MARSHALL L. MICHEL, JUNIOR (*Surgery*, August, 1953) state that meconium peritonitis is a non-bacterial foreign body and chemical peritonitis occurring during intrauterine or early neonatal life as a result of an abnormal communication between the bowel contents and the peritoneal cavity. As meconium is said to reach the ileo-colic junction at the fourth month and is present in the rectum of a five-months-old fœtus, it would seem that intra-partum meconium peritonitis cannot occur earlier than the fourth month. Intra-partum meconium peritonitis is not necessarily fatal, and evidence of it has been shown at operation or at autopsy for other conditions. However, with post-partum meconium peritonitis contamination with bacteria soon occurs, and a fatal outcome is the rule. As regards causes, the authors quote Ramos, who classifies the causes into two groups. Group 1 includes those cases in which there is impediment of progress of bowel contents along the alimentary tract with perforation proximal to the obstruction; this includes intussusception, volvulus, atresia, extrinsic congenital bands, congenital intraabdominal hernia, inspissated meconium and other disorders of a similar nature. Group 2 is characterized by localized defects in the bowel wall, and includes trauma, diverticuli, Meckel's diverticulum *et cetera*. The ætiology of meconium peritonitis is discussed fully, with the arguments for and against the question of trauma and its relationship, and also the occurrence of bowel rupture in the absence of ascertainable obstruction. The diagnosis of meconium peritonitis is predicated on marked distension, which may be present immediately after or at birth, vomiting of a persistent nature and, frequently, cyanosis with difficult respiration. Obstipation is the rule, but occasionally a meconium passage may occur. X-ray examination is diagnostic. Dilated loops of small intestine may point to obstruction, areas of calcific density suggest fœtal peritonitis, and there is free air in the peritoneal cavity, best shown in the upright position. Lipiodol or barium studies are rarely necessary or desirable. The treatment depends upon the nature of the cause. Atresias with perforation are treated by resection of the atresia with anastomosis around the area, care being taken that an associated stenosis is not overlooked. If the obstruction is due to meconium ileus, enterostomy is performed with flushing of the bowel with saline and then 5% pancreatin solution, and closure of the enterotomy and the rupture. When the perforation is apparently spontaneous and no cause can be found at surgery, simple closure can be expected to be attended by the survival of the infant. The authors review eight cases of survival of infants with meconium peritonitis after surgery and add the records of a new case.

Child Training with Social Implications.

F. T. ZIMMERMAN AND BESSIE BURGEMEISTER (*Am. J. Psychiat.*, August, 1953) state that a child's behaviour is a reflection of his emotional adjustment, and this is a reflection of his prior training. Attention is therefore focused on training. The problems of the retarded-child family are not different from those of the normal family except in degree. Parents have been led to believe that such behaviour as temper tantrums and destructiveness is an integral component of mental retardation and so have failed to apply proper training methods. It is considered that the social animal needs to "belong" and will compromise his egocentric wants in order to "belong" and be accepted by society. Growing and learning proceed together, and children can as easily "grow into" bad behaviour as "grow out" of it. Rejection by the parent of the backward child is frequently demonstrated by various mechanisms: excessive concern by one parent, recognizing the child's faults in the other parent, is usually a result of self-blame, with a tendency to compensate in the other direction by the other parent. Numerous parent-child mechanisms of rejection have been observed: (*a*) overt hostility, (*b*) neglect of affection while attending to physical needs, (*c*) excessive training "to improve his mind for his own good", (*d*) excessive affection, so that every expression of the child is accepted, there is no discipline and no order, and no help can be accepted, (*e*) overprotection by parents who need to dominate and control the child's development—evidenced by reluctance of the parents to have the child go anywhere without them. Usually both parents and children need retraining, something more difficult than just training. It is suggested that excessive individualism and "progressive methods" of training create a false position, but are acceptable to the disinterested parent. Conditioned behaviour commenced in the early formative childhood years is recognized as a psycho-physiological principle. It is suggested that the balance between social adaptability and individual preference can best be achieved by a combination of reward and punishment. Unless these are consistent, frustration and neurotic behaviour can be expected. Parents should be indoctrinated in the guiding principles to be used; with these facts they alone can determine the handling of any particular situation. A programme based on love and reason alone must fail, because the child, like the animal, is egocentric; he will demand more and more love, but the reasoning is too difficult to follow. Conditioning is achieved by simple repetition. An outstanding difficulty apparent to the authors is that parents as a class lack a guiding principle or yardstick of behaviour themselves. This lack of inner conscience and softness grows out of excessive permissiveness. The end-result is the spiritual and political vacuum.

Development of Congenital Dislocation of the Hip.

E. W. SOMERVILLE (*J. Bone & Joint Surg.*, November, 1953) states that it is difficult to be certain when congenital dislocation of the hip occurs. It is probably a gradual process spread over a considerable time, the stage of subluxation being passed through before dislocation is finally established. But whether it develops before or after birth is uncertain. There seem to be only two certain factors: (i) that since the head of the femur and the acetabulum are formed together, the head is at first in the acetabulum; (ii) that if complete dislocation is going to occur, it will be present before the age of two years. In 32 hips which have been explored the limbus (*labrum glenoidale*) was found to be inverted into the joint in so-called typical congenital dislocation of the hip, and evidence is presented suggesting that this inversion is the result of an antero-superior dislocation, the result of extension of the hip in the presence of lateral rotation, aided and abetted by anteversion. If the force producing the inversion persists long enough, the limbus will not again evert. Based on the constancy with which the limbus is inverted into the joint in a typical congenital dislocation of the hip, a hypothesis is presented which suggests that the sequence of events leading to established dislocation is: (i) lateral rotation aided and abetted by anteversion, (ii) extension of the hips causing subluxation, (iii) dislocation and inversion of the limbus, (iv) secondary changes in the upper end of the femur, pelvis and acetabulum, which will also develop if the deformity does not progress beyond a subluxation.

Hæmolytic Streptococcal Infection in Childhood.

L. A. RANTZ, M. MARONEY AND J. M. DI CAPRIO (*Pediatrics*, November, 1953) describe the clinical manifestation of streptococcal infection during the first eight years of life. They state that serial studies demonstrated that mild or inapparent infections were frequent

in the first four years of life. In the more severe form of infection the onset was insidious with little or no fever, rhinorrhœa was frequent, anæmia might be produced and the course was protracted with quite frequent suppurative complications. Penicillin therapy, of at least a week's duration, was dramatically effective. A slight and unsustained antibody response was found in this group. After the fourth year of life streptococcal infections became progressively more acute with sudden febrile onset, sore throat, exudative tonsillitis and a self-limited course. Rheumatic fever was observed following these infections only in children above the age of four years. The response to penicillin was less in this group. A sustained rise in antibody formation was the usual immunological response found in these older children. The hypothesis that the changing patterns of clinical response to hæmolytic streptococci are the result of repeated infection by these organisms, rather than due simply to maturation, is considered to be supported by the evidence.

Urticaria Neonatorum.

H. V. L. FINLAY AND J. P. BOUND (*Arch. Dis. Child.*, October, 1953) have made a study of *urticaria neonatorum*. They state that 92% of their cases occurred in the first four days of life. Affected babies showed no constitutional disturbance attributable to the rash, which faded within one to two days. The commoner, simple type consisted of areas of erythema in which a small weal was centrally placed. Less frequently a "pustular" type of lesion, tending to occur more in the flexures, was associated. These "pustules" contained masses of eosinophile leucocytes and never progressed to bulla formation unless secondary infection occurred. The authors cite the repeated opinions in the literature that the rash is of allergic origin, but they consider its cause to be quite obscure.

ORTHOPÆDIC SURGERY.

Ætiology of Traumatic Osteoporosis.

B. DE BURDEAUX AND W. J. HUTCHISON (*J. Bone & Joint Surg.*, April, 1953) performed experiments on dogs to determine whether or not venous stasis produces osteoporosis and to compare this type of osteoporosis, if produced, with that of disuse and trauma. The authors found that osteoporosis of disuse and osteoporosis of stasis vary considerably in time and mode of onset, and that each presents a characteristic microscopic picture. They point out that among the normal stimuli to bone production are stress and strain. These are eliminated in disuse, which leads to a decrease in the rate of bone production, with a normal rate of bone destruction. Therefore osteoporosis of disuse is a passive process and does not appear for several weeks or months in humans; the authors were unable to produce it in less than thirty days in experimental animals. Bone absorption occurs largely in the endosteum and Haversian canals with minimal subperiosteal bone absorption. Osteoporosis of stasis, on the other hand, is an active process with an increased rate of bone absorption and a normal rate of bone production. It occurs rapidly, within several days. Microscopic examination shows that bone absorption is confined largely to the subperiosteal region, and there is no enlargement of Haversian canals. The authors conclude that the osteoporosis which rapidly appears distal to a fracture is largely due to stasis. They believe that disuse plays little part in the production of the osteoporosis frequently seen in the foot and ankle and following a tibial fracture; X-ray evidence of disuse osteoporosis is seen only after many months of immobilization, as has been shown both experimentally and clinically. Also the intensity of bone absorption is too great to be due to disuse. Even after a year or more of disuse there is seldom the degree of bone absorption seen a few weeks after the fracture. The character of acute osteoporosis following trauma differs from that of disuse osteoporosis, in that the former is irregular and involves predominantly the portion of bone distal to the trauma. Osteoporosis of disuse is homogenous and diffuse, involving proximal and distal portions alike.

Adrenocortical Response in Bone and Joint Operations.

J. A. NICHOLAS AND P. D. WILSON (*J. Bone & Joint Surg.*, July, 1953) present results of a study of eosinophile cell levels in 177 patients; 52 were healthy persons used as controls, and 125 were patients who were suffering from various disabilities or injuries of the skeletal system and who underwent different surgical procedures. An eosinophile cell count only was used as an indicator of the improved metabolic effects taking place in the body which depend chiefly upon the response of the adrenal gland to the stimulus of injury. The authors state that these studies confirm previous observations that after operation there normally occurs a profound drop in the eosinophile cell level, which is generally followed by a rise to normal or above normal within three or four days. During the period of eosinopenia the patient is ill, and improvement parallels the rising eosinophile cell counts. Persistent eosinopenia beyond the usual stress period is suggestive of developing complications, either of wound healing or of a general nature. Infections, whether due to pyogenic or to tuberculous organisms, cause stress and resultant eosinopenia. As infection is overcome, the counts rise toward eosinophilia. A rising eosinophile cell count generally parallels a falling sedimentation rate. Eosinophilia when the clinical course of the condition is unsatisfactory is indicative of adrenal insufficiency and should be checked by the Thorn test. A negative result confirms this diagnosis and calls for a trial of cortisone in therapeutic doses. Patients with fresh fractures uniformly showed eosinopenia or evidence of stress. When the fractures had been reduced and fixed, there was a rapid rebound to eosinophilia. Immobilization in plaster encasements did not produce evidence of stress. When plaster jackets were employed for correction of deformity, as in scoliosis, the patients showed eosinopenia. Patients with painful conditions, such as acute low-back pain, exhibited signs of stress. Decrease in the eosinophile cell count did not occur during anæsthesia. Patients in whom acrylic prostheses or large metallic appliances were employed showed persistent eosinopenia lasting as long as five or six weeks. Patients with deformities of congenital origin or with deformities due to poliomyelitis who were admitted for elective surgery showed normal eosinophile cell levels. Patients with cerebral palsy or neoplasms of the bones or joints showed eosinopenia upon admission to hospital.

Capsular Changes and Osteoarthritis of the Hip.

G. C. LLOYD-ROBERTS (*J. Bone & Joint Surg.*, November, 1953) has made the following observations based upon an operative and histological study of 25 osteoarthritic hips subjected to arthroplasty or arthrodesis. In no case did infective, rheumatoid or ischæmic factors determine the onset or complicate the condition. Dissections were made of fresh cadavers to establish the normal structure and function of these tissues at different ages. Fragments of bone and cartilage were found beneath the synovial surface in 23 of the 25 cases of osteoarthritis. The author states that the source of these fragments is the degenerate articular surfaces. The fibrosis of the synovial membrane and capsule follows the synovial hyperplasia which accompanies the phagocytosis of these fragments. A similar histological picture has been produced by injecting fragmented cartilage into the knee joints of rabbits. The injected fragments are found beneath the surface, and synovial hyperplasia is followed by subsynovial fibrosis. The greatest amount of this joint debris is found in the lowest part of the joint cavity. The joint capsule is particularly sensitive to traction. All parts of the capsule are tight in extension, which is the weight-bearing position. Fibrotic shortening of the capsule in the lowest part of the joint cavity explains many of the symptoms and signs of the disease: pain is caused by an attempt to stretch the capsule; muscle spasm occurs in the muscles supplied by the sensory nerves of this part of the capsule; extension, medial rotation and abduction, which tighten this area, are lost first; progressive shortening causes deformity in the opposite direction—namely, flexion, lateral rotation and adduction; the loss of extension causes a more rapid wearing of the articular cartilage on weight bearing; subperiosteal new bone is formed on the undersurface of the neck of the femur.

Tuberculosis of the Elbow.

J. N. WILSON (*J. Bone & Joint Surg.*, November, 1953) has studied 33 elbows in 31 patients with tuberculosis of the elbow. The disease was commonest in the second and third decades. Tuberculosis of the elbow is classified into four types distinguishable radiologically—namely, synovial, extraarticular, coronoid and massive. The author believes that treatment is predominantly conservative. Operation is sometimes indicated for extraarticular lesions. Arthrodesis is advisable in selected cases, but it is not essential for healing. Of 20 patients observed for five years or more, 17 returned to work, seven required permanent splintage and five had residual pain or sinuses. It is suggested that the best position for fixation of the elbow is 30° below the right angle.

Special Article.

NOTES ON TRAUMATIC SURGERY IN KOREA.[1]

THESE notes are designed to present outstanding impressions of traumatic surgery as seen in Korea. They are the outcome of the service of Lieutenant-Colonel K. J. J. Dorney, D.S.O., M.S., F.R.A.C.S., R.A.A.M.C., in this area during 1953, and his association with Allied surgeons and research teams in the battle zone. While many of these impressions were available in the past it is considered that the results of further application during three years in Korea of the lessons of World War II will be of value.

RESUSCITATION OF BATTLE CASUALTIES.

Resuscitation involves the assessment and correction of any defects caused by tissue damage. The high velocity missile produces destruction far beyond its immediate pathway, as each tissue cell is displaced violently at right angles to the missile's path. All missiles, bullets, metal fragments, glass *et cetera* produce the same general effect to a varying extent. While there may be a small wound of entrance and a small wound of exit, the damage to the intervening tissue, as in the liver or gastrocnemius, may be of large and extensive proportions.

Results of Local Tissue Destruction.

Local tissue destruction may be followed by (i) mechanical defects which threaten life or function, (ii) bacterial contamination, (iii) hæmorrhage and shock.

Mechanical Defects.

Mechanical defects may affect a vital function, as in an open wound of the chest, a penetrating wound of the heart with cardiac tamponade, or respiratory obstruction. Here emergency treatment may determine the immediate fate of the patient.

Bacterial Contamination.

Bacterial contamination was universal in a series of 100 patients who were examined approximately three and a half hours after wounding. Clostridia were present in two-thirds of the wounded and *Clostridium perfringens* in one-third. In spite of this, life-endangering infection except in patients with renal insufficiency has scarcely been seen. Gas gangrene has occurred in only one in a thousand cases. Tetanus has not been seen at all. These results have been brought about by the early use of antibiotics—patients receive 300,000 units of penicillin and tetanus toxoid at the battalion aid station (R.A.P.)—and to early and adequate surgical intervention, which consists of exploring each fascial plane widely beyond the edges of the entrance wound, with removal of dirt, cloth, metal and fragmented tissue, and excision of devitalized tissue. This procedure, *débridement*, requires but a short time, as against the natural process, sloughing, which takes several weeks.

The importance of the short time lag between wounding and arrival at a surgical centre is again stressed.

Hæmorrhage and Shock.

Loss of blood is the factor which above all determines the immediate fate of the patient. Hæmorrhage may cause shock very early, whilst shock due to muscle, bone or organ trauma is of more gradual onset.

The reaction of the autonomic nervous system to lowering of the available blood volume is to produce vasoconstriction, and the relation of this vasoconstriction to the lowered blood volume may produce three syndromes as measured by the blood pressure: (*a*) the hypertensive response, (*b*) compensation, (*c*) decompensation.

Hypertensive Response.—The syndrome of hypertensive response is characterized by a blood pressure of 150 to 160 millimetres of mercury (systolic), sometimes rising to 240 millimetres, and is associated with severe pain and usually only moderate loss of blood as in fractures of long bones. It is almost certainly an excessive noradrenaline response. A sharp drop in pressure follows the use of ganglionic blocking agents. These hypertensive patients must be treated with caution as there are two dangers: (i) With the onset of anæsthesia the vasoconstriction mechanism is frustrated, and the blood pressure may fall to low levels very rapidly. (ii) They belong to one of the two categories of patients who may develop anuria; this appears to be due to spasm of the afferent arterioles of the glomerulus, and vasodilators have no demonstrable effect on this spasm. The treatment of these patients is sedation and absolute rest with conservative treatment of blood loss until the blood pressure has become stabilized.

Compensation.—With loss of circulating blood up to 25% (1250 mils) normal blood pressure may be maintained temporarily by vasoconstriction. This is associated with deepening of the respirations and increased heart rate. However, such blood lost must be replaced as soon as possible. Active or passive movements such as travelling over rough roads or even transference to an X-ray machine may have a deleterious effect on the patient, especially if associated with changes of posture. Anæsthesia, by blocking the compensatory response, may convert incipient shock into profound shock.

Decompensation.—With a blood volume deficit more than 25% the body is unable to maintain a normal blood pressure and is in a state of decompensated shock. Decompensation may occur early owing to excessive blood loss or later in the presence of other factors—long-continued anoxia of the vital centres due to decreased circulating blood volume or continued loss of blood plasma into the traumatized area. Cold and the movement of patients by transport ambulance over a long period of time increase this hazard. Decompensation may be corrected by blood transfusion, and if the vasoconstrictor mechanism is still functioning, the blood pressure must be elevated to normal. In Korea, over a series of 4500 battle casualties in which the average time of evacuation was three and a half hours, there was no patient who, in the absence of continuing hæmorrhage or damage to the central nervous system, could not be resuscitated to a stage of normal blood pressure prior to anæsthesia. Massive transfusion was often necessary to achieve this, 15 pints of blood in twenty-four hours being not unusual.

Resuscitation was not always possible after anæsthesia, and the longer the period of severe hypotension, the more blood was needed to resuscitate the patient, as anoxia had a progressively deleterious effect. Furthermore, the group which suffered from severe hypotension for any considerable length of time before transfusion was one of the two classes likely to develop anuria in the post-operative period. This condition of decompensation may progress to the irreversible stage, which does not respond to transfusion and has been seen in Korea following anæsthesia or after a period of severe hypotension lasting more than six hours, because the vasoconstriction mechanism may have failed owing to continued anoxia of the vasomotor centre, or the heart may be unable to respond to increased venous filling pressures owing to prolonged deficient circulation in the coronary arteries.

Two observations were made from a study of 30 patients who on admission had a blood pressure of zero: (*a*) The amount of blood required for resuscitation gives a better indication of the prognosis than the admission blood pressure. (*b*) Mortality is much higher with severe abdominal wounds than with severe extremity wounds. This is not due to difficulty in control of hæmorrhage alone, as the blood volume may be adequately replaced and patients still die of shock or anuria. There are some other factors present and they are possibly associated with irritation or damage of the splanchnic nerves.

Problems of Resuscitation.

The problems of resuscitation will be considered (i) forward of the surgical centre, (ii) at the surgical centre (pre-operative and post-operative resuscitation) and (iii) in the post-operative phase and convalescence.

Resuscitation Forward of the Surgical Centre.

Early evacuation and surgery will save many lives, but restoration of blood volume early should take precedence over these, and no patient should be evacuated until his blood pressure can be maintained at 100 millimetres (systolic). The case of uncontrollable hæmorrhage is the only exception to this. The responsibility of the regimental medical officer or medical orderly is to control hæmorrhage and to restore the blood volume as far as possible after the man has been carried out of the line of fire.

If the blood volume deficit is less than 25%, this can be corrected with one of the plasma substitutes. The more severely wounded men are maintained with dextran throughout the line of evacuation to the surgical centre, where blood is used, and it is here that the helicopter has

[1] These notes have been made available by the courtesy of Major-General F. Kingsley Norris, Director-General of Army Medical Services.

undoubtedly saved many patients who would not otherwise have reached the surgical centre alive. For this reason it is regarded by many as the greatest single medical advance since World War II. On movement the patient does not suffer any trauma likely to prejudice his condition, and the time-saving factor is very great.

The reduction of hæmatocrit readings by the use of plasma substitutes does not cause concern, as a man may be carried through an operation with a hæmatocrit reading of 25% without deleterious effects. On the other hand reduction of blood volume by 50% shocks the patient to such an extent that he will die if not treated.

Morphine.—It has been found that morphine cannot be at present separated from other vasodepressor material substances by assay, and it is believed by some that it may have a deleterious effect by interfering with the vasoconstrictor mechanism. The contention is made that, as pain instead of aggravating shock may actually assist the patient by increasing vasoconstriction, for the alleviation of pain limbs should be firmly splinted, and if morphine is used at all that it should be used only in small doses (one-eighth and one-sixth of a grain). However, most people would still hold that large doses of morphine are necessary in shocked patients to allay pain and restlessness and to prevent unnecessary movement.

Absorption of Fluids.—Studies with deuterium given both orally and subcutaneously show very little absorption into the blood-stream in severely wounded patients. This is due to the intense peripheral vasoconstriction. There is inhibition of the whole activity of the gastro-intestinal tract, and hot sweet tea and other fluids given orally not only are not absorbed but may be vomited during evacuation or anæsthesia. Penicillin given intramuscularly is only poorly absorbed in shocked patients as demonstrated by blood penicillin levels taken at a surgical centre three and a half hours later. It is therefore advised that severely wounded patients receive morphine and penicillin intravenously, as by this route these are absorbed as quickly in shocked patients as in normal people.

Resuscitation at the Surgical Centre.

Stabilization of blood pressure does not take place for some time after a blood pressure of 100 millimetres of mercury (systolic) has been reached, and operation must await this stage. Operation on a patient with a rising blood pressure which may still be under 100 millimetres (systolic) is a hazardous procedure.

Rate of Infusion.—Massive amounts of blood can be given very rapidly in these patients without evidence of overloading the circulation. Those with open chest wounds are an exception, and here care must be taken in transfusion. Those with central nervous system injuries also require only small amounts of blood, as lowering of blood pressure is probably due to injury to the vasomotor centre rather than to blood loss.

Vasoconstrictors.—Vasoconstrictors are of use only in the operative stage when the vasoconstrictor response has been abolished by anæsthesia and also post-operatively for a period of forty-eight hours. "Levophed" and nor-adrenaline have been the main vasoconstrictors used in the Korean War.

Use of Tourniquets.—Some patients with bilateral traumatic amputations are difficult to resuscitate until a physiological amputation has been performed with a tourniquet and further loss of fluid into the traumatized area has been prevented. Surgery can then be performed at leisure providing the circulating blood volume is maintained.

Intraarterial Transfusion.—Intraarterial transfusion may be used with benefit: (a) for the moribund patient who is admitted with an imperceptible pulse and blood pressure and probably six to eight respiratory movements per minute, and (b) in cases of massive hæmorrhage in which a response as evidenced by a rising blood pressure cannot be obtained or is only very slowly obtained by intravenous transfusion. Either the radial artery or the posterior tibial artery may be used, and blood may be given at a very rapid rate, for example, six pints in twenty minutes. There is rapid filling of the arterial trees, and blood passes into the coronary arteries with subsequent stimulation of the cardiac musculature.

Treatment Behind Surgical Centres.

Most surgical patients can be evacuated forty-eight hours after surgery provided that the transportation is not of a damaging nature. Those with abdominal wounds at this stage offer few electrolytic or fluid balance problems, and if these develop they can be treated better in a more static situation.

Plasma Volume Expanders.

In the treatment of shock due to hæmorrhage and trauma, whole blood is the therapeutic agent of choice. Its supply, however, is limited, depending upon adequate numbers of donors. The use of blood is generally restricted to those situations in which transfusions are constantly required, such as hospitals, army surgical centres *et cetera*. It cannot be stockpiled against an emergency, as under present conditions blood becomes unsuitable if stored longer than three weeks.

There is no complete substitute for whole blood, but there are several efficient agents for restoring plasma volume and maintaining blood pressure. The term "plasma volume expander" is applied to these.

Pooled human plasma, either in the wet form or in the dry form which can be readily reconstituted with an equivalent volume of sterile water, is reasonably stable, and can be satisfactorily stored and transported. The use of large donor pools is associated with a definite incidence of infection with homologous serum jaundice, which both ultraviolet irradiation and chemical treatment have so far failed to eliminate. This risk may be reduced by fractionation or by using small donor pools.

A solution of the albumin fraction of plasma performs in many respects as effectively as plasma and contains the essential constituent for maintaining plasma volume, is free of the virus of serum jaundice and remains in circulation longer than any of the present synthetic plasma volume expanders. Furthermore, fractionation of whole plasma yields other products of importance to civilian and military medicine—such as fibrinogen, prothrombin and γ globulin.

Human albumin is supplied in either 5% or 25% solution, the latter in the form of 100 ml ampoules, each of which is theoretically the osmotic equivalent of 500 mls of blood. In practice, however, the blood volume is not increased to this extent, probably because the wounded are usually dehydrated to a certain degree.

Since World War I, an efficient substitute for plasma capable of economic production and prolonged storage has been sought. Such a substitute when in solution should have the following physical properties: it should have a sedimentation rate, viscosity and osmotic pressure comparable to those of plasma, it should be free from pyrogenic, allergic and toxic properties, it should be subject to slow tissue metabolism but not prolonged tissue storage, and it should have a molecular structure sufficiently large to prevent elimination by the kidneys before the patient has manufactured enough blood cells and plasma proteins to restore his own blood volume.

Several substances possess many of these properties and they can be divided into three classes: (i) Proteins. Serum albumin and gelatine are in this class. (ii) Carbohydrates. Gum acacia and pectin, derived from citrus fruits, were used in World War I, but were subject to prolonged tissue storage. Dextran is one of this class. (iii) Synthetic polymers. Polyvinyl pyrrolidone is produced by chemical combination of formaldehyde and acetylene. This substance does not cause as prompt or as great a rise of blood pressure as the others.

Dextran and gelatine are the plasma expanders most used in Korea. They are comparable in the length of time they remain in the circulation and in their effect in maintaining blood pressure.

Dextran is a polysaccharide composed of glucose units and is produced by the action of a coccus *Leuconostoc mesenteroides* on sucrose. It is produced in varying molecular sizes and a 6% solution with a molecular weight of approximately 70,000 is now being used in Korea. This solution is storable and transportable, has no toxic properties, and is as effective as plasma or albumin in the immediate restoration of blood volume and blood pressure. There are limitations in providing by means of dextran for long-continued resuscitation of the patient during evacuation, as this substance is fairly rapidly excreted in the urine; in some cases only 40% is present in the plasma on the patient's arrival at a hospital three hours after infusion. Repeated observation of the patient's condition is essential if helicopter evacuation is not available.

Dextran is also used as a resuscitative agent during surgery and post-operatively, and a series of metabolic studies were carried out on nine severely wounded patients under these conditions. When the blood pressure was stabilized after operation, each patient was given a transfusion of one litre of dextran at the rate of 250 mils hourly,

and plasma and urinary dextran estimations were made at regular intervals. Plasma dextran concentrations dropped rapidly in the first twelve hours with rapid excretion in the urine. Of the total dextran infused 40% was in the bloodstream immediately after infusion, after six hours 20% was present, after twelve hours 14% and after seventy-two hours 1% or 2%; 46% appeared in the urine in the first six hours. Approximately 30% of the administered dextran could not be accounted for and was probably metabolized or lost into the wound.

It was found that casualties with a plasma volume deficit of 500 to 1000 mils could be maintained on dextran alone. The hæmatocrit reading falls about 4·5% with each 500 mils of dextran, and after an infusion of three to four pints care must be taken, as acute anæmia may develop.

When blood is available in limited quantities, the use of both blood and dextran in equal quantities has proved to be a useful adjunct to therapy.

Gelatine is derived from the collagen of bone and skin degraded by several methods; 3% modified gelatine in 0·7% sodium chloride solution was used. Similar metabolic studies were carried out as for dextran. When one litre of gelatine solution is infused into a severely wounded man over a four-hour period, 50% remains in the plasma at the end of the infusion, 30% is excreted in the urine and 20% is lost into the wound, or the extravascular compartment. Six hours after completion of the infusion only 20% remains in the plasma, 65% being excreted in the urine.

These two substances are the most effective agents used at present. If a higher molecular weight could be achieved they would have a more prolonged and enhanced effect. In the United Kingdom carbohydrate plasma volume expanders have been developed with a molecular size of 120,000 or more. They are not blood substitutes and are useful only in that they may enable a patient with blood loss to be evacuated in reasonable condition to a situation where blood is available, or they may be used in combination with blood during resuscitation, surgery and the post-operative period.

Primary Repair of Arterial Wounds.

Previous wars have demonstrated that approximately 1% of all battle casualties suffered damage to a major artery. Furthermore, of all amputations performed on battle casualties, 20% were the result of direct vascular injuries. A study to assess the feasibility and value of primary repair of arterial wounds was therefore undertaken. A series of 60 unselected casualties were considered.

In this series a major artery was ligated in only two patients, as neither could be resuscitated sufficiently to permit a major operation. In the first patient ligation of an axillary artery resulted in an ischæmic viable extremity. In the second, popliteal ligation was followed by gangrene of the lower extremity. With these two exceptions the patients reported represented a succession of unselected casualties. Minor arteries, especially those distal to the popliteal and brachial arteries, were frequently ligated, since the sacrifice of such a single vessel rarely endangers the extremity.

The operation of election was *débridement* and direct end-to-end anastomosis of the vessel. Three patients had small arterial defects, less than 25% of the circumference of the vessel, which permitted transverse repair. In all other instances it was necessary to excise a complete cross-section of the artery before attempting anastomosis. The defect was so large in three patients that an autogenous vein graft was necessary.

The primary wound was enlarged and explored and the vascular defect located. Both the proximal and distal ends of the severed artery were debrided to a point approximately one centimetre into apparently normal vessel. Such a criterion is admittedly grossly empirical. The artery was freed proximally and distally so that approximation could be made without tension; collateral circulation was sacrificed if necessary. The adventitia was stripped from the cut ends of the vessel to facilitate suture, and finally the clamps were momentarily released from the vessel ends to remove any residual clots. Anastomosis was performed with a continuous everting suture of 5 (00000) fine silk. Vascular clamps of the Potts principle were used throughout this work. When direct anastomosis could not be performed without tension, an autogenous vein graft was inserted. The cephalic or saphenous vein was the most frequent site used for the source of the graft. In all repairs care was taken to cover the anastomosis or graft site with muscle.

Arterial spasm in the proximal segment of the severed artery offered a technical problem in only five instances. In each case a sponge soaked in 2·5% papaverine solution was wrapped around the vessel, care being taken to cover the most proximal area of spasm. The sponge was left in place for fifteen to twenty minutes and then removed.

Anticoagulant therapy was not used and would have been fraught with danger. After completion of the vascular repair, any devitalized muscle was excised, skin and subcutaneous tissues were left open, and the extremity was flexed to 20° and immobilized in a split case. Antibiotic therapy was used as a routine. Sympathectomy was not carried out.

Results.

The results of primary repair are shown in Table I.

TABLE I.

Artery.	Number.	Type of Repair.			Result: Number of Subsequent Amputations.
		Anastomosis.	Vein Graft.	Transverse Repair.	
Common carotid	3	2	1	0	0
Subclavian ..	1	0	1	0	0
Axillary	7	5	2	0	0
Brachial	16	15	1	0	0
External iliac ..	2	2	0	0	1
Common femoral	2	2	0	0	0
Superficial femoral	13	10	3	0	2
Popliteal ..	14	12	0	2	3
Posterior tibial ..	2	1	0	1	1
Total.. ..	60	49	8	3	7

Comparison of these results with results of ligation in previous wars demonstrates beyond question the value of primary repair.

TABLE II.

Artery.	Number of Patients.	Number of Amputations After Primary Repair.	Amputation Rate in World War II. (Percentage.)
Axillary	7	0	43
Brachial above profunda	4	0	55
Brachial below profunda	12	0	25
External iliac	2	1	46
Common femoral	2	0	81
Superficial femoral ..	13	2	54
Popliteal	14	3	72

Several of the comparisons in Table II are statistically insignificant, but the overall comparison is conclusive. Such a study, in which amputation is the only end-result to be considered, fails to demonstrate the full value of primary repair. After ligation, a very ischæmic functionally insufficient extremity frequently results. This syndrome has not yet been seen after primary anastomosis.

Long-range follow-up is essential for complete evaluation, but the analysis of results to date permits several clinical observations.

Early operation is very desirable. Lapse of time from injury to repair varied from three to forty hours with the results shown in Table III.

The average time lag was 8·3 hours.

Secondary hæmorrhage was observed only once. In this instance a secondary vein graft was successful.

The seven failures of primary repair resulted from two causes: (i) irreversible damage sustained pre-operatively from ischæmia, (ii) errors in operative technique.

In five of the seven failures direct anastomosis (4) or vein graft (1) was carried out eight or more hours after injury. In three of these cases, in which repair was performed ten, thirteen and sixteen hours after injury, subsequent amputation below the anastomosis revealed gangrenous muscle, but an actively spurting arterial circulation. In these three, it would appear that irreversible damage to the muscle had occurred prior to the reestablishment of arterial continuity.

In other cases a shorter time lag or a better collateral circulation prevented these changes.

The second factor leading to failure—namely, technical errors—centres primarily around the tension of the anastomosis and to an almost equal extent around an inadequate *débridement* of the artery and surrounding tissues. In at least three instances thrombosis occurred at the suture line; two of these were associated with multiple (2) anastomoses at different levels of the same artery, and tension was the cause of failure.

TABLE III.

Time Lag Before Repairs. (Hours.)	Number of Patients.	Number of Amputations.
0 to 3 ..	3	0
4 to 6 ..	22	2
7 to 9 ..	19	1
10 to 12 ..	9	2
13 to 15 ..	5	1
Over 15 ..	2	1
Total ..	60	7

The seventh patient had a severe wound infection associated with devitalized muscle.

One patient died on the second post-operative day from an associated cerebral injury. This case was included in the seven failures already described.

Conclusion.

In the management of battle casualties the treatment of choice of arterial injuries is primary repair by direct end-to-end anastomosis or by autogenous vein graft. Although the proportion of successful anastomoses decreased after eight hours, the success of an individual anastomosis cannot at present be predicted on the basis of time lag.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Alfred Hospital, Melbourne, on September 9, 1953.

Extensive Hæmangioma of the Lower Limbs.

DR. JOHN BEGG described the case history of a male child, aged three and a half years, who had first reported to the children's surgical out-patient department of the Alfred Hospital in May, 1951. At that time his mother's only complaint was that he had extensive birth marks on his right buttock, thigh and leg.

On examination of the child multiple cavernous hæmangiomatous lesions were found scattered over the right lower limb and in the perineum. He was considered then as unsuitable for surgery, but, as there had been some hæmorrhage from the lesions, he was referred for radiotherapy. He next reported in November, 1951, because of intermittent attacks of pain and disability in the affected leg. In the attacks, which came on suddenly, he would cry with pain, refuse to use the limb and resent any handling of it. The attacks lasted a period varying from a few hours to several days, when they would wear off and he would run around as usual. When seen in one attack, the leg was held flexed on the thigh, although the knee could be straightened with considerable difficulty and pain. The hæmangiomatous mass on the inner side of the knee appeared to be tender. No effusion could be demonstrated in the joint. In June, 1953, he developed a swelling on the postero-medial aspect of the right knee. This presented the characteristic signs of a semimembranosus bursitis. An investigation of the vascular system of the right lower limb and pelvis was undertaken by the Alfred Hospital radiological department. Although filling of the angiomatous masses with diodone was obtained, no evidence of intrapelvic or intraspinal abnormality of the vascular system was detected.

Dr. Begg said that there seemed to be no doubt that the recurrent attacks of pain and disability in the right lower limb were associated with the presence of the angiomata, but the exact mechanism producing them remained unexplained at present. It was well known that angiomata in the region of the knee joint were occasionally associated with intraarticular lesions of a similar nature, the nipping of which produced severe pain, but the absence of any demonstrable effusion in the joint did not favour such an hypothesis. It was possible that intramuscular angiomata, which probably existed in the present case, could have produced painful prolonged cramps by interference with the blood supply of skeletal muscle; but, if so, it was strange that with such a comparatively common lesion it was not mentioned in the literature more often.

In conclusion, Dr. Begg said that it was proposed to remove surgically the enlarged semimembranosus bursa and the tender angiomatous mass on the inner side of the knee joint. At the same time opportunity would be taken to inspect the interior of the joint.

DR. RUSSELL HOWARD said that he had seen only one other similar case. The present patient had no osseous involvement, and there appeared to be no contraindication to surgery. Dr. Howard thought that surgery would relieve the pain and give a good result.

DR. F. DOUGLAS STEPHENS said that as the arteriogram had shown normal major vessels in the limbs it would not matter how much cavernous tissue was removed. Sufficient should be removed to alleviate the trouble.

Spontaneous Pneumopericardium.

DR. G. RALEIGH WEIGALL presented the case histories of two patients with spontaneous pneumopericardium who had been admitted to hospital within a few weeks of each other. They were presented because of the rarity of the condition, few references to similar cases having been found in the literature, and also because of the absence of any demonstrable cause for the extravasation of air in the situations in which it was found.

The first patient, a tall thin boy, aged twelve years, of poor physique, with no relevant previous history, had been admitted to hospital on November 15, 1952, with a history of cough and malaise for three days. Thirty hours before his admission he had complained of pain in the left side of the chest after a severe coughing bout, but was able to go to school. On the same evening in bed he complained of severe pain in the left shoulder and neck, and his breathing became very distressed. He was brought to hospital four hours later. His temperature was 99° F., his pulse rate was 120 per minute with good volume, and his respiratory rate was 24 per minute. He was quiet and comfortable while propped up in bed, and was not cyanosed.

On examination of the boy his trachea was found to be deviated to the right, and there was surgical emphysema above the left clavicle and in the left side of the neck. There was diminished respiratory movement on the left side of the chest, which was hyperresonant to percussion. The breath sounds were diminished on the left side, and fine râles were heard over both lungs. The apex beat was in the fifth left intercostal space inside the mid-clavicular line. The sounds were regular and clear. His electrocardiogram was normal, and a Mantoux test result was negative. No other abnormality was found. No treatment was given except rest. Dr. Weigall said that the diagnosis was established, and as subsequent progress of the child was recorded radiologically to the time of his recovery, the skiagrams to be demonstrated would tell the whole story. Five weeks after recovery from this illness the child had developed lobar pneumonia of the middle lobe of the right lung, from which he made a quick recovery. He had attended the out-patient department subsequently and was perfectly well.

The second patient was a boy, aged six years, who had been subject to asthma for two years and had been treated by Dr. C. E. Sutherland with injections of protein extracts and potassium iodide. He had contracted measles three weeks before admission to hospital, but he was not very ill and had no more than a mild bronchitis, which cleared completely. Soon afterwards, following an upper respiratory infection, he developed mild asthma. Loud expiratory and inspiratory rhonchi were heard over both lungs, but he was not more distressed than he was usually with such attacks until, after a bout of coughing, he developed dyspnœa and cyanosis with a short distressing cough. He was admitted to hospital, and examination revealed resonance over the whole of the left side of the chest. Breath sounds could be heard over the upper part of the left lung; but at the

left lung base the breath sounds were diminished appreciably, and coarse râles only were audible. On the right side of the chest, loud breath sounds and expiratory and inspiratory rhonchi were heard. The apex beat was palpable in the fifth left intercostal space, half an inch inside the mid-clavicular line. The sounds were regular and clear.

Dr. Weigall said that when he examined the boy he diagnosed left pneumothorax. There was a doubtful history of his having had the condition once before, but that could not be confirmed. He was admitted to hospital urgently, and on admission his temperature was 98·4° F., his pulse rate was 110 per minute, and his respiratory rate was 28 per minute. He was cyanosed and very distressed, but after oxygen and atropine, grain 1/150, had been administered he soon improved and was comfortable within half an hour. A skiagram showed air in the pericardium but not in the pleural space. By the following morning, breath sounds were heard over the upper lobe of the left lung, and his chest was clinically clear. His progress was uneventful from then on. Dr. K. Newman Morris carried out a bronchoscopic examination a week later and found no abnormality.

Dr. Weigall said that subsequently the boy had suffered from several more attacks of bronchitis and asthma, but he had not caused any further anxiety. There were no other signs or symptoms beyond that of ordinary mild bronchitis and asthma, and the attacks were easily relieved by "Ephedrobarbital". Dr. Weigall thought the child must have had a temporary block in a bronchus from a plug of mucopus and relief of the spasm by atropine and oxygen enabled him to cough this out without sequelæ in the lung, but only after some air had been forced into the pericardium and mediastinum. It was difficult to explain the means by which this occurred. Dr. Weigall said that the boy's progress would be better appreciated from serial skiagrams.

DR. H. A. LUKE described the radiographic features of the patients whose clinical histories had been given by Dr. Weigall. Serial reports were as follows:

Case I.

14/11/52: P.A. & Lateral Chest. There appears to be a thin layer of air within the pericardial sac, and associated with this there is some air present in the upper mediastinum, particularly anteriorly. The air in the pericardial sac extends over the left side of the heart and passes anterior to, posterior to and inferior to the heart, but cannot be demonstrated over the right side of the heart. There is no evidence of any other abnormality of the heart, mediastinum or lung fields. There is no evidence of foreign body. A little subcutaneous emphysema is demonstrated in the neck.

17/11/52: There has been partial absorption of the air in the pericardium and the mediastinum. There is still no evidence of any abnormality in the lung fields.

18/11/52: Heart screening and barium swallow—contour of heart and cardiac movement appear normal. Œsophagus appears normal.

19/11/52: There is still a little air present in the anterior mediastinum, but most of the air in the region of the pericardium has been absorbed. Air is visible only in the lateral film.

21/11/52: Further absorption of air in the mediastinum. No evidence of air in the pericardium.

Five weeks later, films showed an area of pneumonic consolidation in the right middle lobe, which quickly cleared. The last film taken in February, 1953, three months after the original examination, demonstrated normal heart outline and lung fields.

Case II.

9/2/53 and 10/2/53: There appears to be a little air present in the pericardial sac, particularly posteriorly and inferiorly, and there is also evidence of mediastinal emphysema, with some air tracking up the soft tissues in the left side of the neck. The air in the pericardial sac is only faintly visible in one of the P.A. films, although more clearly visible in the lateral projection. Fusion of the right first and second ribs present. An accessory lobe appears to be present at the right base, otherwise the lung fields are clear.

11/2/53: There has been almost complete absorption of the air in the pericardial sac. The lung fields remain clear.

17/2/53: Heart outline, mediastinal shadow and lung fields now appear normal.

Commenting on the radiographic findings, Dr. Luke said that the two patients demonstrated the spontaneous appearance of air in the mediastinum and in the pericardium in small quantities following some degree of respiratory embarrassment, in one case during a bout of coughing, and in the other during an attack of bronchial asthma. Both were young male subjects, one thirteen years and the other six years old, and both suffered a moderately severe central chest pain. In the first patient there was a larger quantity of air present than in the second, and the air was absorbed over about seven days, whereas in the second patient the air was absorbed more rapidly.

A search of the literature showed that a few similar cases had been described. In 1939 R. Graebner had described six cases of spontaneous pneumopericardium following obstructive laryngitis. In five of these a tracheotomy had been performed before the recognition of the pneumopericardium. The sixth patient, a girl of fifteen years, was similar to those in the cases described by Dr. Weigall. In 1942 Trimble *et alii* had described a case of spontaneous pneumopericardium in a patient with apparently arrested pulmonary tuberculosis. Unfortunately several other references appearing in the *Index Medicus* were unobtainable in Australia, and many others obviously should have been described as cases of hydropneumopericardium or pyopneumopericardium.

Dr. Luke said that it was of interest to mention that Hannan in his original description of spontaneous pneumomediastinum did not refer to the possibility of air entering the pericardial sac. The mechanism of entry of air into the pericardial sac was unknown. However, two possible methods had been described in the literature, namely, rupture of the sac and air embolism.

DR. H. B. KAY said that he was interested in the fact that the heart sounds were not noisy, as usually occurred in cases of pneumopericardium. The fact that air was present in the pericardial sac probably did not matter, as air never caused tamponade, in contrast to hæmopericardium, in which tamponade was the rule. Dr. Kay said that he presumed that the air entered the pericardial sac through some defect in the pleuro-pericardial reflection.

DR. V. L. COLLINS said that in his experience of pneumomediastinum air was not detected in the pericardial sacs, and in no case had he heard noisy heart sounds. The only occasion on which he could recall having heard the typical loud heart sounds was in a case of pyopneumopericardium, the result of infection with gas-forming organisms.

DR. HOWARD WILLIAMS said that he had met with only one case of pneumopericardium, and in that the classical pinging heart sounds were present.

DR. B. L. DEANS said that it was important to differentiate air in the pericardial sac from air between the mediastinal pleura and pericardium. In one case, as seen in an oblique film, air seemed to follow the pulmonary artery to its bifurcation into right and left branches. That seemed to confirm the fact that air was actually in the pericardium.

DR. H. MCLORINAN agreed with Dr. Collins that pneumopericardium bore a close relationship to mediastinal emphysema. If more of the latter cases were carefully examined it was possible that pneumopericardium might be detected more often, for example in children with pertussis or in cases of laryngeal obstruction with tracheotomy and violent coughing. Dr. McLorinan said that he was interested in Dr. Kay's statement that air in the pericardial sac was harmless. Probably the symptoms in such patients were due to air in the mediastinum or pleural cavity.

Dr. Weigall said that Dr. Trumble had put forward the theory that pneumopericardium was due to a defect in the ceiling of the pleuro-pericardial reflection.

Dr. Luke remarked that recent information had disclosed that air injected presacrally for the purpose of outlining the retroperitoneal areas could track up to the mediastinum or pericardio-phrenic space, and lead to pneumomediastinum. It would seem reasonable, during examination of films of patients who had been given presacral injections of air, to look a little higher than the lumbar regions.

Patent Ductus Arteriosus.

DR. J. M. GARDINER presented two patients with patent *ductus arteriosus*. In both cases the classical continuous murmur was absent. The diagnosis was established by cardiac catheterization, and subsequently confirmed at operation. (The case reports of these children are being published in detail elsewhere in THE MEDICAL JOURNAL OF AUSTRALIA.)

DR. M. L. POWELL said that he was surprised at the history of maternal rubella in both patients whom Dr. Gardiner had presented, and at the high incidence of maternal rubella in the series of patients with patent *ductus arteriosus* at the

Alfred Hospital. At the Royal Children's Hospital surgery had been performed on 65 children with patent *ductus arteriosus*, and another 20 children were on the waiting list. Only one of those children had a history of maternal rubella. At the Royal Children's Hospital no cardiac catheterizations had been done. The diagnoses were made clinically. Of the 65 children whose ducti had been tied, only one had no continuous murmur, but that child had all the other clinical features, including a high pulse pressure, and radiological evidence of a large heart with congested lung fields. The decision to operate had been based on clinical findings, and a patent *ductus arteriosus* had been found at operation. Dr. Powell said that it was commonly thought that in infancy the murmur of a patent *ductus arteriosus* was purely systolic. At present he was observing nine infants less than eighteen months old who had typical continuous murmurs. All had big hearts and urgently required operations. That was contrary to the usual teaching that a continuous murmur should not be expected until the infant was two years old.

DR. RUSSELL HOWARD said that he was surprised that Dr. Officer Brown did not divide the big *ductus arteriosus*. He was certain that in some cases it required division. A big ductus was a short ductus, and the procedure of ligating in three places put a great strain on the ductus itself, the pulmonary artery and the aorta, and bleeding often occurred. Dr. Howard said that he remembered one case in which a large ductus had been tied once only. The patient died soon afterwards from a dissociated illness, and at necropsy the ductus was found to be patent. A large ductus should be divided, and tying without dividing should be restricted to the small ducti.

DR. H. B. KAY said that the atypical group was interesting. A large *ductus arteriosus*, especially if associated with cardiac failure, should be subjected to early surgery. It was commonly said that a ventricular septal defect was associated with a small aorta, and a patent *ductus arteriosus* with a large aorta, but that was not so in many cases, and investigational procedures were often necessary. Some patients with patent *ductus arteriosus* had mid-diastolic murmurs and were sometimes diagnosed as suffering from mitral stenosis. The diagnosis of patent *ductus arteriosus* was sometimes incorrect, and it was unwise for practitioners to give an unequivocally bright prognosis to parents on the results to be expected from surgery.

DR. M. L. POWELL expressed the opinion that once the diagnosis of patent *ductus arteriosus* had been made, surgery should not be delayed if practicable. The prognosis was much better in childhood, and it was difficult to prophesy what the state of the patient's health would be in ten years' time.

Dr. Gardiner, in reply to Dr. Powell, said that when there was a history of rubella and a congenital heart defect, a large proportion of the patients concerned turned out to have patent *ductus arteriosus*. That was not to say that a large proportion of patients with patent *ductus arteriosus* had a history of maternal rubella; it merely said that when confronted with a congenital heart deformity and a history of maternal rubella one tended to think of patent *ductus arteriosus*. Dr. Gardiner said that he was very interested to hear of Dr. Powell's large series of babies with continuous murmurs. There was little doubt that many infants had systolic murmurs only. All the infants in one series from an American source had systolic murmurs alone. The diagnosis of patent *ductus arteriosus* could be very difficult on occasions and the condition could be confused with a patent interventricular septum and an aorta-pulmonary septal defect.

Clinical Diabetes Insipidus.

DR. JOHN FARRER presented the case history of a boy, aged six years, who had been admitted to Prince Henry's Hospital in January, 1953, for investigation of what appeared to be *diabetes insipidus*. The initial stage of his investigations was performed at Prince Henry's Hospital under the direction of Dr. T. D. Hagger and subsequently he was transferred to the Royal Children's Hospital, where he was investigated in the Clinical Research Unit by Dr. W. B. Macdonald.

The child's parents were first cousins. The family history was not relevant and there was no record of abnormal thirst or polyuria. The patient was the only child. There was no hyperemesis, renal disorder or febrile illness during the mother's pregnancy, and her labour was normal. The baby was artificially fed from birth. His birth weight was six pounds two ounces, and at twelve months of age he was of expected weight. Feeding was somewhat difficult and culminated in forcing. Subsequently the feeding difficulties persisted, and the patient now ate very little meat and eggs, had his vegetables strained, and at the age of six had been "off the bottle" for only a year. There had been very little additional vitamin intake since infancy. He had at no time developed a craving for salt. His weight gain since the age of twelve months had been slow, especially for the past two years, so that at the age of six years he weighed thirty-six and a quarter pounds and was three feet seven and a half inches in height. His milestones, apart from feeding and micturition, were passed at normal intervals, and at the present time he was a little above average intelligence as estimated by his mother. Bowel habits had never caused concern, but he had never grown out of nocturia, therefore presenting with the main complaint of enuresis. The first cause for concern occurred at the age of four and a half years, when he was noticed to be running to the tap for a drink frequently. A urine test at that time showed no abnormality. At the age of six years he was thin and underweight, suffering from thirst and polyuria with enuresis. Examination of the urine revealed no albuminuria, glycosuria or casts. The specific gravity of his urine was 1·002 and after twelve hours of water deprivation was still 1·002. A fluid balance chart was instituted for a few days, followed by administration of 0·5 cubic centimetre of "Pitressin" tannate in oil daily for fourteen days. However, his fluid balance was not affected and the specific gravity of his urine was between 1·001 and 1·002. An excretion pyelogram was normal. Results of Wassermann and Kahn tests were negative. Skiagrams of the pituitary region, long bones and chest revealed no abnormalities. He was then transferred to the Royal Children's Hospital for more specific evaluation of his renal function.

Dr. Farrer concluded by submitting four main questions for consideration: Firstly, was the condition congenital or acquired or a certain percentage of both? Secondly, if it was congenital, was it a manifestation of recessive characteristics brought about by close relationship in marriage or was it a chance occurrence? Thirdly, if it was acquired, was it a renal infection, for which no evidence had been found, or was it a metabolic defect imposed on him by faulty diet and/or management as a whole? Fourthly, if it was acquired, by what means was the whole or part of the condition reversible in the interests of the future management of the patient?

DR. W. B. MACDONALD discussed the investigations which had been carried out at the Royal Children's Hospital. He said that there was no doubt that the child had polyuria and thirst, although those symptoms were by no means as intense as one would have expected from a subject of *diabetes insipidus*. After drinking one litre of water the child commenced to excrete urine almost immediately despite injections of five units, 10 units and 20 units of "Pitressin" on three separate occasions. Within four hours of each injection the volumes of urine excreted were 830 millilitres, 490 millilitres and 592 millilitres respectively. Other "Pitressin" effects were observed, such as pallor, increase in blood pressure, and colicky abdominal pains. Other tests demonstrated that there was an impairment of renal function. There was no abnormality of the urine found on microscopic examination or ward testing on numerous occasions, and urine chromatography performed by Dr. J. Perry showed no excess of amino acids. After eighteen hours' water deprivation the urine specific gravity was 1·007 and the urine urea concentration was 885 milligrammes *per centum*, but the child showed no clinical evidence of dehydration. However, serum electrolyte estimations made before and after water deprivation demonstrated a distinct difference. The hæmoglobin value rose from 9·8 to 11·3 grammes *per centum*, the hæmatocrit from 29% to 32%, and the serum protein content from 6·90 to 8·4 grammes *per centum*. The blood urea concentration rose from 48 to 72 milligrammes *per centum*, the serum sodium concentration from 140 to 153 milliosmols per litre, and the serum chloride concentration from 102·5 to 115 milliosmols per litre. The other serum electrolyte concentrations did not appreciably change, and the blood pH stayed at 7·44. There was an undoubted water loss despite deprivation.

Renal clearance studies showed a definite impairment of glomerular function. Corrected to surface area of 1·73 square metres, endogenous creatinine clearances corrected for chromogens were 35·0 and 38·8 millilitres per minute, and urea clearances at a rapid rate of urine flow were 33·5 and 31·3 millilitres per minute. Phenolsulphonphthalein excretion was impaired, only 37% of dye being excreted in four hours and 49% in six hours. Urine ammonia concentration was poor, never being above five milliequivalents per litre, although serum bicarbonate concentrations were normal and there was no change in blood pH. There was a high urinary concentration of sodium and chloride, but the significance of that was hard to assess in the absence of balance studies, which were not done.

Dr. Macdonald said that faced with this evidence of impairment of renal function one had to decide what its origin was. There was nothing in the history or on examination of the urine to suggest either glomerulonephritis or pyelo-

UNIVERSITY OF MICHIGAN LIBRARIES

nephritis. Congenital renal hypoplasia might have accounted for the syndrome, but skiagrams demonstrated anatomically normal kidney size, and excretion pyelograms prepared at Prince Henry's Hospital and at the Royal Children's Hospital showed no distortion of the pelves. The last pyelogram showed a distinct diminution in the intensity of dye shadow, and that served to confirm poor tubular excretion of dye as shown by the phenolsulphonphthalein test.

The fact that symptoms did not develop until the child was over four years of age suggested that the functional derangement was the result of a progressive disorder and was a factor against a diagnosis of congenital "Pitressin"-resistant *diabetes insipidus* first described by Waring, Kajdi and Tappan. One possibility which had to be seriously considered was that the child was suffering from a metabolic disorder such as cystine storage disease (Lignac-Fanconi disease), in which polyuria, polydipsia and "Pitressin"-resistance were features. The kidneys in cystine storage disease became atrophic and fibrotic, finally resembling those of chronic pyelonephritis, even in the absence of infection. The child under discussion showed no evidence of dwarfism, and skiagrams of his bones were normal. Only one chromatographic examination of his urine had been done. That showed no abnormality, but, according to Bickel, that need not negate the diagnosis, as amino aciduria could be inconstant and discrete. A slit-lamp examination of the child's corneas had been made but no abnormality was detected.

The position at the moment with the child was that the exact origin of his renal lesion was unknown. There was no doubt that he had a renal lesion, and the "Pitressin"-resistant polyuria probably was due to a combination of an intrinsic tubular defect and a progressive degenerative process. Dr. Macdonald said that despite the lack of positive evidence he considered that a bone marrow puncture to seek for cystine crystals was desirable, especially as Dr. Perry had nicely established the diagnosis by that means in another child with cystinosis recently.

Dyggve and Samsøe-Jenson from Copenhagen had described one boy, aged twelve years, almost identical with the present child, even to the approximation of urea and inulin clearances. They too were left in the predicament of being unable to find the origin of the "nephropathia".

Dr. Macdonald concluded by answering the questions which Dr. Farrer had submitted. He said that it was impossible to state on the available evidence whether the condition was congenital or acquired, but he suspected, as mentioned, that it would turn out to be a congenital metabolic disorder like cystine storage disease which was progressing insidiously to renal failure. The consanguinity of the parents supported that theory, as in several cases reported in the literature on cystinosis there was a history of consanguinity. There was a chance that the renal dysfunction was the result of a static congenital abnormality, but the child's long-term prognosis was not very good in Dr. Macdonald's opinion. There was no indication for medicinal treatment at present, but the child's protein intake had been kept to basic metabolic requirements, so as to limit osmotic diuresis.

DR. H. N. B. WETTENHALL said that a child who presented with symptoms of polydipsia and polyuria required careful investigation. It was necessary to exclude a urinary tract infection, but it was becoming more commonly realized that metabolic disorders such as Fanconi's syndrome had to be seriously considered. An apparently uncomplicated urinary tract infection required more thorough investigation if a recurrence developed. Dr. Wettenhall quoted the case of a child seen recently at the Royal Children's Hospital as a sufferer from an apparent urinary tract infection. More detailed examination revealed that the underlying disorder was cystine storage disease. It was a rare disease; but if it was not suspected the diagnosis would not be established.

DR. ROBERT SOUTHBY inquired whether a renal biopsy was justified. Skiagrams had excluded the more obvious anatomical deformities, but as the child's prognosis was uncertain at present, a renal biopsy might provide valuable information. Dr. Southby said that possibly renal biopsies would be useful prognostic aids in other chronic renal disorders.

Dr. Macdonald, in reply, said that he would not care to advise needle biopsy on a small child, and it would appear reasonable to postpone decision on laparotomy and biopsy until a bone marrow puncture had been made.

DR. T. D. HAGGER said that the investigation of the child illustrated the value of cooperation between hospitals.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

RIVER POLLUTION.[1]

RIVER POLLUTION . . . is a filthy noisome nuisance, and an abomination to many places in the interior. Defiling one of God's best and greatest of all luxuries and blessings, viz., good, pure water . . . is a prolific source of engendering disease and mortality. . . . The stench arising from the dead bullocks so frequently met with on our public highways, and which contaminates and pervades the air for miles around with almost unendurable, abominable smells, is bad enough, but to pollute God's streams and rivulets of heaven-distilled water with sheep-washing is enough to breed plagues and pestilence throughout the length and breadth of the land. Cleanliness, it is said, is next to godliness. Yes, but forsooth, how much respect is paid to cleanliness or sanitary laws in these colonies; when one man may . . . poison, in this manner, a whole city or community with the utmost nonchalance? Is it not worse than arrant mockery to say that the people of New South Wales are governed by enlightened statesmanship, when such hotbeds of disease as these are tolerated in our midst? Of petty state peddling, the rankest of selfish ignorance, there is enough, but of a knowledge of sanitary laws, there is, indeed, but very little. No wonder our virgin soil is already cursed with grasshoppers, blight, weeds, burrs, thistles, plagues, and pestilences innumerable, while tyrannical selfishness and truculent conduct exercises its unlimited sway and behest. It is very well to preach missionary schemes, distribute Bibles amongst the heathen, &c., but it would sound far more natural to doff the sacerdotal garb, and tell the apeing, pretending Christian, that "uncleanliness is a sin" . . .

Special Correspondence.

NEW ZEALAND LETTER.

BY OUR SPECIAL CORRESPONDENT.

Report on Hospital Reform.

IN June, 1953, the Government appointed a Consultative Committee on Hospital Reform under the chairmanship of Mr. H. E. Barrowclough (recently appointed Chief Justice of New Zealand as Sir Harold Barrowclough, P.C.). Its report has just been released by the Minister of Health, the Honourable J. R. Marshall, and has been the subject of mostly favourable comment by the Press. Action in the matter is required within the next two years, as by that time the abolition of rates levied on local bodies as a source of hospital revenue will be complete, and the funds will come solely from Government. The present system of hospital boards, of many years' standing, was based on essentially a local responsibility for finance. The committee bases its plan on central ministerial responsibility.

Amongst the measures advised are the diminution in number of hospital boards from 37 to 23, and the giving of some appointed seats to the larger boards, the remainder being still to be elected by popular vote. In Dunedin and Auckland there is to be university representation. There are to be five regional authorities, based on the four main centres and Palmerston North, with an overall duty within their regions. Coordination between the regions, and certain central functions of standardization and research, are left with the hospital division of the Health Department. Urgently needed are studies on, and the introduction of, a system of costing for the various sections of hospital work. Greater aid for private hospitals is advised as a less expensive method of maintaining beds than new building in public hospitals.

[1] From "Typhoid Fever, Its Pathology and Treatment, Based on Rational Physiological Principles", Sydney, 1873, by Andrew Ross, M.D., medical practitioner, of Molong, New South Wales.

In comment it has been said more than once that the regional conception is welcome, but that, as far as the report goes, there is insufficient definition as to scope and powers of, and the various relationships between department, regional authority and local hospital board. Doubtless much would depend on the calibre of men secured for the full-time executive posts, and the part-time membership of the regional authorities. Though there has been considerable discussion of (and in certain quarters support for) the idea of a corporation or commission to take the place of the department as a coordinating body between the four, five or six regions, no suggestion to this effect was made by the committee.

In general the report is a useful stimulus to thought in hospital affairs, and action by the Government (perhaps not in 1954, election year) is attended with great interest.

Correspondence.

THE COLLEGE OF GENERAL PRACTITIONERS.

SIR: The first annual report of the College of General Practitioners has been received with interest and enthusiasm in England. The introduction of a National Health Scheme in Australia which is favourable to the general practitioner has not lessened the need for such a faculty here. A planned scheme of post-graduate study for general practitioners is inevitable if academic efficiency is to be maintained or advanced. Nor is it less necessary to hold—or dare I say restore—the ethical standards of the profession. As a third point, the faculty might well help in strengthening the hand of the Federal Council in medical politics. Let us hope that all eligible practitioners will join the College. By so doing they will not only show an interest in their own welfare, but will help to make secure the medical life of their successors. The freedom in general practice which we have thus far enjoyed may still be the heritage of our younger brethren.

Yours, etc.,

William Street,
Bellingen,
New South Wales.
February 15, 1954.

MERVYN E. H. ELLIOTT.

PERFORATION OF THE ŒSOPHAGUS AND RUPTURE OF THE AORTA DUE TO A PIECE OF FLY-WIRE.

SIR: While reading the report of a case of "perforation of the œsophagus and rupture of the aorta due to a piece of fly-wire" by J. H. W. Birrell and W. A. Syme in your issue of February 13, 1954, I was struck by some points of similarity to a report published by Ashleigh Davy and myself (M. J. AUSTRALIA, January 14, 1950).

This prompts me to inquire whether the circular dark area in the centre of the lower half of their photograph, shown traversed by a dotted line, may not be a false aneurysmal sac, separating the œsophagus from the aorta. The authors speak of the œsophageal rent as "being in direct continuity with the laceration in the aorta", but describe the latter as "one centimetre in its greatest diameter", while the former "was a large rent some three centimetres in maximum diameter". Where then is the direct continuity? May it not be that an aneurysmal sac lies in between?

I would like to suggest that the process to which Dr. Birrell and Dr. Syme tend to attribute a subsidiary role, that is, "Ulceration and rupture would, of course, be hastened or facilitated by contact of the pulsating vessel with a pointed foreign body", may be the initial fundamental lesion. In other words, it may be the early piercing of the aortic wall by the sharp point rather than "the formation of an abscess in the tissue surrounding the œsophagus and large vessels" which sets in train the sequence of events leading to death. If this be true, extravasation of blood may follow the piercing of the aortic wall, a false aneurysmal sac may be formed, and later—helped undoubtedly by infection—the œsophageal wall may be eroded.

My point in suggesting this possible pathological process is not to start a contentious correspondence, but to offer a practical approach to treatment. I wonder whether the interval after the initial perforation of the aortic wall may offer a prospect for thoracotomy and repair of the perforation, small as it is. Antibiotic therapy, œsophagoscopic examination and removal of the foreign body do not seem capable of effecting a cure.

Yours, etc.,

143 Macquarie Street,
Sydney,
February 22, 1954.

VOLNEY BULTEAU.

THE TREATMENT OF ACUTE POLIOMYELITIS.

SIR: May I congratulate Dr. Gordon Colvin and Dr. S. E. J. Robertson on their excellent articles on acute poliomyelitis published in the journal of January 16, 1954? Might I have space to comment on, and stress certain points:

1. The extreme danger of "working off the sluggish feeling of the early stages", as Dr. Colvin puts it. I think practically all paralysis would be avoided if we could diagnose the disease at this stage.

2. In the first two to three weeks after the onset of meningitic symptoms, all active muscular movement should not only be avoided but forbidden, and the patient not allowed to lift even a finger to do anything for himself. This must be stressed on patient, sisters and nurses.

3. Passive movements at any time—no active movement before four weeks, and then only very little before six weeks, and then increasing as rapidly as possible.

4. Dr. Robertson says "pain can be relieved by aspirin or codeine". Might I make another plea as I have done before—mental distress, excitement, insomnia and intense pain are almost unbearable. Aspirin and codeine are useless alone—anyway, in adults. In addition, give morphia, phenobarbital, potassium bromide, chloral, "Carbrital", or all if necessary, or combinations of same, and they may be needed for six weeks. Nothing is so demoralizing as pain and/or lack of sleep. Is the modern young doctor afraid of drugs, other than antibiotics?

Yours, etc.,

Gerrale Street,
Cronulla,
New South Wales.
February 27, 1954.

A. M. MACINTOSH.

Obituary.

ALEXANDER JUETT.

THE following tribute to the late Dr. Alexander Juett has been sent by Dr. R. D. McKellar Hall.

Alexander Juett died on May 19, 1953, at the age of sixty-six years, after a long illness. Elected to a Rhodes Scholarship, he went up from Christian Brothers College, Perth, to Brasenose, Oxford, to read medicine. He was placed in the second class in physiology in 1910, elected to a Hulme Research Scholarship in 1911 and qualified B.M., B.Ch. in 1913 after three years at the Middlesex Hospital. He became head of the Government Hospital at Nannine, Western Australia, on his return to Australia in 1913 and married in the next year. He served in the Australian Army Medical Corps throughout the war, was mentioned in dispatches and was badly gassed in France. At the end of hostilities he was lieutenant-colonel in charge of the medical side of the Repatriation Department in Fremantle. On demobilization he commenced practice in Perth, and thereafter steadily became one of the most prominent, and certainly one of the most selfless, orthopædic surgeons in Western Australia. He was the pioneer orthopædic specialist in the State and had to face the opposition customary in those days. This opposition he steadily overcame without creating enmity, and it was not long before his colleagues recognized his superior merits. He initiated the orthopædic clinics at both the Children's and Perth Public Hospitals, and at the time of his death was consulting honorary orthopædic surgeon to both. He was a foundation Fellow of the Royal Australasian College of Surgeons and a foundation member of the Australian Orthopædic Association. He was a consultant with the rank of major to the Australian forces in the second

UNIVERSITY OF MICHIGAN LIBRARIES

World War. Every year he personally conducted the medical examination of Rhodes Scholar candidates in Western Australia, and among other organizations—and these were widespread—to which he insisted on giving his services free was the Fairbridge Farm School at Pinjarra. He was honorary surgeon to the Maimed and Limbless Ex-Servicemen's Association and took a great personal interest in each individual member who needed his attention. All this while he was himself a constant sufferer from the after-effects of being gassed. He was a genuinely unselfish man. He is survived by a widow.

ALEXANDER HAMMETT MARKS.

DR. ALEXANDER HAMMETT MARKS, who died recently in Brisbane at the age of seventy-four years, was the son of a distinguished Queensland physician, the Honourable C. F. Marks, M.D., M.L.C. He was educated at the Brisbane Grammar School and at Trinity College, Dublin, from which he graduated M.B., B.Ch., B.A.O. in 1902 and proceeded to the M.D. degree three years later.

Returning to Brisbane in 1904, he took a full part in the medical life of the fast-growing city and was at different times honorary radiologist, junior physician and junior and senior gynæcologist at the Brisbane General Hospital, and also a member of the honorary staff of the Lady Bowen Hospital. He was a Foundation Fellow of the Royal Australasian College of Surgeons. He served on the Council of the Queensland Branch of the British Medical Association from 1919 to 1927, having been President in 1914, and was President of the Medical Defence Society of Queensland from 1931 to 1946. He was always interested in the welfare of Nurses and was President of the Australasian Trained Nurses' Association from 1923 to 1934. He retired from active practice in 1945, but continued his work as senior medical officer to the Australian Mutual Provident Society until 1950.

Dr. Marks had a distinguished record of military service. He enlisted in 1914 as regimental medical officer in the Third Field Infantry Brigade, with which he served throughout the Gallipoli campaign. Later he became deputy assistant director of medical services of the Fourth Division in Egypt and in France, where he was awarded the Distinguished Service Order in 1917. He commanded the Second Field Ambulance, then formed the Seventeenth Field Ambulance, and later commanded the Second Casualty Clearing Station from 1917 to 1918, winning the Croix-de-Guerre. In 1918 he became Assistant Director of Medical Services of the First Division and was made a Commander of the Order of the British Empire for meritorious services. After the war he was appointed Deputy Director of Medical Services of the First Military District and held this post from 1921 until his retirement in 1937.

Dr. G. W. Macartney writes: Almost forty years ago I met Dr. Alex. Marks at Victoria Barracks whilst examining recruits for the First Australian Imperial Force. Then began a friendship which neither time nor change diminished.

This faculty for friendship was one of his outstanding qualities. He was a friend as well as a doctor to all his patients, many of whom owed him not only their health but much material help as well.

His happy disposition and keen sense of humour, inherited from his Irish ancestors, made him a most lovable character; always younger than his years, he showed an interest in guiding and developing his younger contemporaries during his period as Deputy Director of Medical Services of the First Military District, and his choosing and encouraging of the later generation for posts in the Army Medical Service helped to produce some of the efficiency of the Australian Army Medical Corps in the last war. Nothing pleased him more than to entertain young and old members of the Australian Army Medical Corps and to reminisce, over a smoke and a drink, about different personalities and episodes of the First World War.

His chief hobby was an interest in old furniture, of which he had a great knowledge. During his life he gathered together a valuable collection. Each new piece he obtained had a story connected with it. At the 1950 Australasian Medical Congress he exhibited a consulting room of fifty years ago from his own collection. No mean exponent at the carpenter's bench, he repaired and renovated many pieces thought of little or no value, and the finished article was a craftsman's joy. He was thus able to supply all the *objets d'art* for at least four homes—his own, his son's and his two daughters'. His other hobby was his farm twelve miles from Brisbane, where he spent many happy week-ends and holidays.

He served the British Medical Association as a councillor for many years and was President of the Branch in 1914. His fund of common sense and his ability were a great asset to the Branch. His presidency of the Medical Defence Society for fifteen years is a record, and the present satisfactory position of the Society is largely due to his tact and understanding of human nature.

He was a good friend to the nursing profession over a long life, both as an examiner and as President of the Australasian Trained Nurses' Association for eleven years. The examination howlers of some candidates caused him much amusement, though the students always had his sympathy and help.

A family doctor of the old school, with a bent for obstetrics and gynæcology, he practised in Wickham Terrace for nearly forty-five years, following in the footsteps of his father, the Honourable C. F. Marks, who began practice in Wickham Terrace in 1880. His son now carries on the old family tradition. It is, I think, unique that three generations in one family have carried on the medical profession at the same address in Brisbane for a period of over seventy years; this was a great pride to Alex. Marks, for he regarded tradition as a great factor for good in everyday life.

Alex. Marks received three decorations for war service and held five degrees and diplomas and carried on a busy practice for forty-five years; so he can truly be said to have had a life full of achievement, and he enjoyed every moment of it.

One cannot view the passing of such a man without feeling that the profession and his patients have lost a valued friend and that the community at large is the poorer for his passing.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 7, of February 5, 1954.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointment.—Michael Francis Bennett is appointed Surgeon Lieutenant (for Short Service) (on probation), dated 2nd December, 1953.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointment.—Ben MacMahon Wadham is appointed Surgeon Lieutenant, dated 4th December, 1953.

Promotion.—Surgeon Lieutenant Arthur George Harrold is promoted to the rank of Surgeon Lieutenant-Commander, dated 29th November, 1953.

District Naval Medical Officer.—Surgeon Lieutenant Trevor Nelson Hatfield is appointed District Naval Medical Officer, Western Australia, dated 10th December, 1953.

Royal Australian Naval Volunteer Reserve.

District Naval Medical Officer.—The appointment of Surgeon Lieutenant-Commander Daintrey Ned Kekwick as District Naval Medical Officer, Western Australia, is terminated, dated 9th December, 1953.—(Ex. Min. No. 7—Approved 4th February, 1954.)

AUSTRALIAN MILITARY FORCES.

Regular Army Special Reserve.

Royal Australian Army Medical Corps (Medical).

VX700350 Captain P. J. Ryan is transferred from the Citizen Military Forces and to be Temporary Major, 31st December, 1953.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—1/62652 Captain (provisionally) W. P. Bottomley relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District) in the honorary rank of Captain, 17th December, 1953.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/146603 Honorary Captain B. L. Reid is appointed from the Reserve of Officers, and to be Captain (provisionally), 27th October, 1953. To be Major, 23rd December, 1953: 2/142865 Captain T. H. Lee.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/101812 Captain P. J. Ryan is transferred to the Regular Army Special Reserve, 31st December, 1953.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

1st Military District.—To be Honorary Captains: Daniel Brodie Hogg, 14th December, 1953, and Merton Hugh Thew, 21st December, 1953.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The notification regarding the appointment to a permanent commission of Squadron Leader J. F. Howell-Price (023025) as approved in Executive Council Minute No. 103 of 1953, appearing in *Gazette* No. 71, dated 12th November, 1953, is withdrawn.

Air Force Reserve.

Medical Branch.

The following former officers are appointed to commissions with rank as indicated: (Flight Lieutenant (Temporary Squadron Leader)) P. A. Tod (277138), 10th September, 1953, N. L. Newman (261890), 24th October, 1953; (Flight Lieutenant) W. J. Ferguson (025643), 8th September, 1953, J. F. Marrington (021966), 15th October, 1953, L. E. Goldsmith (264853), 30th October, 1953.

The following are appointed to commissions with rank as indicated: (Flight Lieutenant (Temporary Squadron Leader)) Edmund Muller (277619), 2nd October, 1953; (Flight Lieutenant) N. G. York (431805), 26th June, 1953.

The appointment of Flight Lieutenant R. I. Johns (267898) is terminated, 19th October, 1953.

The notification regarding the provisional appointment of No. A4717 Air Cadet Bernard John Cox as approved in Executive Council Minute No. 102 of 1953, appearing in *Gazette* No. 71, dated 12th November, 1953, is withdrawn.

Australian Medical Board Proceedings.

QUEENSLAND.

THE following have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners: Corbett, Graham, M.B., B.S., 1952 (Univ. Melbourne); Ferguson, John, M.B., B.S., 1953 (Univ. Melbourne); O'Loughlin, Denis Christopher, M.B., B.S., 1953 (Univ. Melbourne).

The following additional qualifications have been registered: Tange, John Damian, M.R.C.P. (London), 1953; Wilson, Chester Gilmore, M.R.C.O.G. (England), 1953.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, as duly qualified medical practitioners: Muir, John David Henry, M.B., B.S., 1953

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 13, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	11(8)	1(1)	..	..	..	..	..	..	12
Amœbiasis	..	..	..	..	1	..	..	..	1
Ancylostomiasis	..	..	..	..	..	..	4	..	4
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(1)	11(11)	2(2)	..	2(2)	..	1	1	21
Diphtheria	3	2(1)	2	..	5(4)	..	..	..	12
Dysentery (Bacillary)	..	..	3(2)	1(1)	2(2)	12(12)	..	..	18
Encephalitis	..	1(1)	..	1	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	20(12)	..	..	7(3)	..	..	..	27
Lead Poisoning	..	..	1(1)	..	..	..	..	..	1
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	2(2)	1(1)	1(1)	1	4	..	..	..	9
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	1(1)	..	..	..	..	1
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	18(12)	4(3)	1(1)	1(1)	11(10)	..	..	..	35
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	5(3)	..	..	8(4)	..	..	..	13
Salmonella Infection	..	..	..	..	1	..	..	..	1
Scarlet Fever	4(2)	21(10)	5(4)	5(3)	1(1)	..	..	..	36
Smallpox	..	..	1(1)	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	32(26)	16(12)	24(9)	4(2)	6(2)	9(4)	..	..	91
Typhoid Fever	1(1)	1(1)	..	..	..	..	..	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	1(1)	1	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

UNIVERSITY OF MICHIGAN LIBRARIES

(Univ. Melbourne); Breen, John Thomas, M.B., B.S., 1953 (Univ. Melbourne); McCulloch, Douglas Neil, M.B., B.S., 1953 (Univ. Melbourne); Perry, George Henry Tasman, M.B., B.S., 1953 (Univ. Melbourne); Spence, Frank Meldrum, M.B., B.S., 1953 (Univ. Melbourne); Townley, Reginald Rugeley Woods, M.B., B.S., 1953 (Univ. Melbourne); Varey, John Forsyth, M.B., B.S., 1953 (Univ. Melbourne); Warden, Frederick George, M.B., B.S., 1953 (Univ. Melbourne); Hale, George Stewart, M.B., B.S., 1953 (Univ. Melbourne); Jackson, William Desmond, M.B., B.S., 1953 (Univ. Melbourne); Lyons, Ashwell Lloyd, M.B., B.S., 1953 (Univ. Sydney); Bierzynski, Henryk, M.B., B.S., 1953 (Univ. Sydney); de Castella, Hubert Cecil, M.B., B.S., 1953 (Univ. Melbourne); Davis, Geoffrey David, M.B., B.S., 1953 (Univ. Sydney); Franklin, Enid Mary de Vere, M.B., B.S., 1953 (Univ. Melbourne); Hawkins, John Graham, M.B., B.S., 1953 (Univ. Melbourne); Hughes, Raymond Maldwyn, M.B., B.S., 1953 (Univ. Sydney); Maxwell, Violet Kathleen, M.B., B.S., 1953 (Univ. Melbourne); Perkins, Graham Paton, M.B., B.S., 1953 (Univ. Sydney); Stephens, David Deloitte, M.B., B.S., 1953 (Univ. Sydney).

The following additional qualification has been registered: Birchall, Ida Lois, F.R.C.S. (Edinburgh), 1938, F.R.C.O.G., 1951.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course for Diploma in Dermatological Medicine.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that, provided five enrolments are received, a course for the diploma in dermatological medicine will be conducted in Sydney in 1954. The Part I course will be held from May 17 to August 6, 1954, and the Part II course from August 17, 1954, to February-March, 1955. Fee for attendance at both courses is £94 10s. Those interested in obtaining further particulars of the diploma or wishing to make enrolment on the course are requested to communicate as soon as possible with the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5238, BW 7483.

Notice.

STOLEN ELECTROCARDIOGRAPH.

NORTH SYDNEY detectives are anxious to recover a Sandbourn model American-make 240-volt electrocardiograph, number 2059, weighing about 35 pounds, in a case, which has a brown cloth material cover with zipp fastener. The electrocardiograph was stolen from a car in the driveway of the home of Dr. John H. Deakin, 7 Prince Albert Street, Mosman, on the night of February 21, 1954. Anyone knowing the whereabouts of the electrocardiograph is asked to contact the North Sydney detectives.

Deaths.

THE following deaths have been announced:

HARTRIDGE.—Oliver Keith Hartridge, on February 20, 1954, at Caulfield, Victoria.

LORENCE.—George Stephen Lorence, on February 27, 1954, at London.

Medical Appointments.

Dr. S. G. Mallarky has been appointed Deputy Director of Pathological Laboratories in the Department of Public Health, New South Wales.

Dr. J. Lisyak has been appointed to the Division of Mental Hygiene in the Department of Public Health of New South Wales.

Dr. B. E. Sharkey has been appointed to the State Hospitals and Homes in the Department of Public Health of New South Wales.

Nominations and Elections.

THE undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Collings, Joseph Silver, M.B., B.S., 1946 (Univ. Sydney), B.Sc., 131 Kilby Road, North Kew, practising at 2 Erin Street, Richmond, Victoria.

Diary for the Month.

MARCH 15.—Victorian Branch, B.M.A.: Finance Subcommittee.
MARCH 16.—New South Wales Branch, B.M.A.: Ethics Committee.
MARCH 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MARCH 17.—Western Australian Branch, B.M.A.: General Meeting.
MARCH 17.—Victorian Branch, B.M.A.: Clinical Meeting.
MARCH 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
MARCH 18.—Victorian Branch, B.M.A.: Executive of Branch Council.
MARCH 23.—New South Wales Branch, B.M.A.: Council Quarterly.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.